No. ____

IN THE

Supreme Court of the United States

JEANNETTE ROGERS DULAN,
Petitioner,

v.

HUNTSVILLE HOSPITAL ASSOCIATION, INC.;
DR. ANDERSHOCK, M.D.,
Respondents.

Petition for a Writ of Certiorari to the United States Court of Appeals for the Eleventh Circuit

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PETITION FOR WRIT OF CERTIORARI

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QUESTIONS PRESENTED

1. Whether the lower court's grant of summary judgment and the panel's per curiam affirmance is contrary to clear U.S. Supreme Court precedent set forth in *University of Tennessee v. Elliott*, 478 U.S. 788 (1986), and other cases, by refusing to give collateral estoppel effect to the uncontested and unappealed findings of the Department of Health and Human Services' Centers for Medicare and Medicaid Services (CMS), which determined that (a) out of twenty emergency room patients it sampled, Petitioner Dulan was the only patient to whom Respondent Hospital failed to give a medical screening prior to requiring that answers be given concerning insurance coverage; (b) the Hospital's conduct violated the Emergency Medical Treatment and Active Labor Act's (EMTALA's) prohibition against delay in providing an appropriate medical screening examination and treatment in order to inquire about the individual's method of payment or insurance status as required by EMTALA and CMS regulations; and (c) "the deficiencies are so serious that they constitute an immediate threat to the health and safety of any individual who comes to the emergency department and requests examination or treatment for an emergency medical condition".

2. Whether the District Court and the Circuit Panel erred by not following the U.S. Supreme Court's decision in *Erie Railroad Company v. Tompkins*, 304 U.S. 64 (1938), by disregarding Alabama Supreme Court precedent which dispenses with the need for Petitioner to have an expert witness in a malpractice case when, as here, Petitioner introduced a recognized standard or authoritative medical text or treatise to prove what the proper standard of care is, by introducing into evidence the ACC/AHA Guidelines which are the universally accepted standards for the initial evaluation and treatment of all patients presenting to an emergency department with chest pain, and further produced uncontested evidence that demon-

strated that neither Respondent Hospital nor its agents met any of those standards.

3. Whether the District Court and Circuit Panel erred by not following the U.S. Supreme Court's decision in *Erie Railroad v. Tompkins*, 304 U.S. 64 (1938) by disregarding Alabama Supreme Court equitable estoppel decisions which, as applied to the facts of this case, provided precedent for disallowing Respondents' surprise motion to strike Petitioner Dulan's identification of expert witness (on timeliness grounds) when Respondents had earlier agreed to depose Petitioner's expert in Washington, D.C.

4. Whether the District Court and Circuit Panel erred in improperly applying U.S. Supreme Court precedents governing summary judgment, which prohibit courts from weighing the evidence and require that courts draw all reasonable inferences in the non-moving party's favor.

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IN THE

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No. ___

JEANNETTE ROGERS DULAN,

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HUNTSVILLE HOSPITAL ASSOCIATION, INC.;
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Petition for a Writ of Certiorari to the United States Court of Appeals for the Eleventh Circuit

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PETITION FOR WRIT OF CERTIORARI

Petitioner Jeannette Rogers Dulan respectfully prays that a writ of certiorari issue to review the judgment of the United States Court of Appeals for the Eleventh Circuit, entered on September 19, 2006 in the above titled proceedings.

OPINIONS BELOW

The two sentence per curiam opinion of the Court of Appeals is not published and is reproduced in the Appendix. (App. B at 17a-24a). The unpublished opinion of the United States District Court for the Northern District of Alabama granting Respondents' motion for summary judgment is reproduced in the Appendix (App. A at la-16a).

JURISDICTION

The Court of Appeals entered its judgment on September 19, 2006. (App. B at 17a). The Court of Appeals denied Petitioner's timely filed petition for rehearing and rehearing en banc on November 7, 2006. (App. C at 19a).

STATUTES AND REGULATIONS INVOLVED

The Emergency Medical Treatment and Active Labor Act (EMTALA), 42 U.S.C.1395dd (a), Medical Screening Requirement, provides, in relevant part:

> In the case of a hospital that has an emergency department, if any individual . . . comes to the emergency department and a request is made on the individual's behalf for examination or treatment for a medical condition, the hospital must provide for an appropriate medical screening examination within the capability of the hospital's emergency department, including ancillary services routinely available to the emergency department, to determine whether or not an emergency medical condition . . . exists.

The Emergency Medical Treatment and Active Labor Act (EMTALA), 42 U.S.C.1395dd (h), No Delay in Examination or Treatment, provides:

> A participating hospital may not delay provision of an appropriate medical screening examination required under subsection (a) of this section or further medical examination and treatment required under subsection (b) of this section in order to inquire about the individual's method of payment or insurance status.

The Department of Health and Human Services, Centers for Medicare & Medicaid Services' (CMS') Revised EMTALA Interpretive Guidelines (May 13, 2004), Part II, The Principal Focus of the Investigation, provides in relevant part:

> CMS will determine if the violation constitutes immediate jeopardy to patient health and safety. The hospital

has the opportunity to present evidence to CMS that it believes demonstrates its compliance and the opportunity to comment on evidence CMS believes demonstrates the hospital's noncompliance. CMS' regional offices retain delegated enforcement authority and final enforcement decisions are made there.

STATEMENT

A. Overview and Summary

Petitioner seeks review of the ruling of the Eleventh Circuit Court of Appeals per curiam affirmance of the United States District Court's grant of summary judgment to Respondents. The District Court erred as a matter of law in failing to give preclusive effect to the final, unappealed, and uncontested findings of CMS. CMS' findings determined, among other things, that (a) out of twenty emergency room patients it sampled, Petitioner Dulan was the only patient to whom Respondent Hospital failed to give a medical screening prior to requiring that answers be given concerning insurance coverage App. D at 21a; (b) the Hospital's conduct violated the Emergency Medical Treatment and Active Labor Act's (App. E at 30a) prohibition against delay in providing an appropriate medical screening examination and treatment in order to inquire about the individual's method of payment or insurance status as required by EMTALA and CMS regulations (App. E at 32a); and (c) "the deficiencies are so serious that they constitute an immediate threat to the health and safety of any individual who comes to the emergency department and requests examination or treatment for an emergency medical condition". (App. E at 33a).

On October 6, 2000, Petitioner Dulan came to Respondent Hospital's emergency room (ER) complaining of chest pain and vomiting. The Hospital's records listed Petitioner as 55 years of age, 15 years postmenopausal, history of high blood pressure, and as having a family history of coronary artery disease. (App. H at 66a-67a). The Hospital's emergency room

physician in charge of Dulan's care, Respondent Andershock, stated at his deposition that Dulan's clinical presentation was consistent with cardiac ischemia. (App. H at 67a). Dulan's myoglobin level showed an elevated level of 75.4; a normal reading is 58. (App. K at 82a). And, it is undisputed that Dulan's initial EKG was abnormal and remained abnormal throughout her stay in the ER (App. H at 73a-74a).

Respondent Huntsville Hospital is a Medicare participating Hospital within the meaning of 42 USC 1395dd(e) of EMTALA. On August 10, 2004, it received a preliminary determination letter of termination from the Medicare provider program of The Centers for Medicare and Medicaid Services of the Department of Health and Human Services (CMS) for violations of EMTALA 42 CFR 489.24, because of its "[f]ailure to avoid delay in providing a medical screening exam in order to inquire about the individuals' method of payment or insurance status for an individual who came to the Hospital requesting services." (App. E at 32a). CMS based its determination solely on the administrative complaint Dulan had filed concerning Respondents' handling of her medical emergency on October 6, 2000. CMS determined that the "Hospital's Emergency Department staff failed to provide (1 of 20 sampled patients) Patient Identifier # 4 [Appellant Dulan] with an appropriate medical screening prior to inquiring about her insurance coverage." (App. D at 21a).

Pursuant to CMS' letter of August 10, 2004, the Hospital was given the option to avoid termination by providing "credible allegation or credible evidence of correction of the deficiencies" (App. E at 33a). CMS also gave the Hospital the option to "successfully prov[e] that the deficiencies found did not exist." *Id.* The Hospital chose to correct the deficiencies rather than mount a defense.

CMS' findings indicate that: Dulan came to ER at 12:16 p.m. (App. D at 20a). Respondent began its registration of Dulan at 1:02 p.m. Dulan continued to express her symptoms

to Respondent at 1:10 p.m (App. D at 25a) and, at 1:35 p.m., complained of general anterior chest pain to her left neck and shoulder. (App. D at 26a). Petitioner's registration did not end until 1:40 p.m. (App. D at 25a). CMS sampled 20 patients and found that the Hospital only failed to give Petitioner Dulan a medical screening prior to inquiring about her medical insurance. (App. D at 21a). Additionally, CMS' findings stated that a person identified as Nurse # 4 stated "that it looks like [Petitioner Dulan] was registered before (she received a medical screening)." (App. D at 25a).

At 2:40 p.m. (App. H at 65a) Respondent's ER physician # 1 saw Dulan. At 7:15 p.m., Respondents transported Dulan to its cardiac care unit. Physician #3 saw Dulan at 7:20 p.m. It was not until 8:30 p.m. that the Hospital sent Dulan to the catheterization lab. (App. D at 25a-26a). Dr. Chen, the cardiologist performing the angioplasty noted in his report that, "unfortunately," Dulan's symptoms were "already nine hours after onset" before appropriate action was taken. (App. D at 26a).

Counsel for Dulan requested access to patient records to obtain patient data to do a statistical analysis of Respondent Hospital's ER. Dr. AnnMaria DeMars, Petitioner's expert, in reviewing the data, found significant racial disparity in Respondent Hospital's administration of aspirin (App. J at 81a) and vigorously defended the applicability of her findings to the facts of this case. *Id.*

The American College of Cardiology and American Heart Association have published guidelines that set forth standards that govern the expected conduct of ER physicians in making decisions concerning patients suspected of having heart attacks or related conditions. (App. G at 40a-63a). Dr. Cooper, Petitioner's expert, stated in his report that the ACC/AHA standards are the "universally accepted guidelines . . . for the initial evaluation and treatment of all patients presenting to the emergency department with chest pain." (App. F at 39a).

Appellees never once stated that this is not the case. Moreover, Respondent-Appellee Dr. Andershock referred to these standards in answering a question during his deposition. (App. H at 67a). The undisputed facts in the record indicate that Respondents failed to comply with the following ACC/AHA guidelines:

(1) The Hospital failed, contrary to the ACC/AHA guidelines, to administer aspirin immediately to Dulan, a patient suspected of suffering from cardiac ischemia. (App. G at 41a and 44a). Instead, the Hospital allowed over two hours to elapse before it gave Dulan aspirin. Respondent's Counsel acknowledged that Respondents only administered aspirin to Petitioner Dulan at 3:10 p.m. according to the medical records. (App. I at 86a). Moreover, Dr. Andershock admitted that Dulan's "clinical presentation was consistent" with "cardiac ischemia." (App. H at 72a). Moreover, Dulan's disparity study indicates that Respondent Hospital discriminates on the basis of race in the administration of aspirin to suspected heart attack victims. (App. J at 76a, 87a).

(2) The Hospital failed to perform an ECG/EKG within 10 minutes as required by the ACC/AHA Guidelines. (App. G at 41a, 43a, 44a). Dr. Andershock's Deposition reveals that the Hospital only performed an ECG/EKG after approximately 65 minutes from the time Petitioner arrived in the ER and 35 minutes after her registration. (App. H at 74a).

(3) The Hospital's attending physician failed to make an initial assessment within 10 minutes as required by the ACC/AHA guidelines. (App. G at 40a-41a). Instead, Dr. Andershock, the attending physician, did not see Dulan until over two hours after her arrival. (App. H at 82a).

(4) The Hospital did not immediately consider reperfusion therapy, as the ACC/AHA guidelines require if the ECG demonstrates ST elevation. (App. G at 41a and 46a). Remarkably, irrefutable evidence of record demonstrates that ST

elevation was clearly printed on the ECG report as part of the highly sophisticated computerized interpretation. Dr. Andershock admits this to be the case. (App. H at 70a). Dr. Andershock also admits that all of Dulan's EKG's were abnormal. (App. H at 73a-74a).

(5) The Hospital failed to perform an emergency angioplasty within no more than 90 minutes or administer a clot-buster within 30 minutes of the patient's arrival in the Emergency Department, as is required by the ACC/AHA guidelines. (App. G at 41a). The clear evidence of record plainly indicates that none of these standards of care was followed and approximately 496 minutes elapsed before the Hospital performed an angioplasty. (App. D at 25a-26a).

B. The Lower Courts' Decisions

1. The District Count's Decision Below

a. EMTALA

Delay Under EMTALA, Section 1395dd (h). The Court recognized that CMS had determined that Respondent Hospital had delayed Dulan's screening to ask her questions about her insurance. The Court stated that the Hospital had acted within the protections of the law (App. A at 11a-12a, n.6) and that CMS's findings of a violation of EMTALA were merely conclusions based on an inaccurate standard. The Court stated that Dulan must establish that Respondent Hospital delayed the screening because of the inquiry. (*Id.*)

b. State Law Medical Malpractice.

The Court stated that Dulan had failed to produce any expert witness to refute Respondents' evidence that the standard of care was met. The Court did not mention that it had struck Petitioner's expert for failure to timely identify Petitioner's expert. The Court also stated that Dulan failed to produce any expert testimony regarding the applicable standard of care and whether Respondents' breach proximately

caused Dulan's injury. The Court held that that since Dulan had not established that she had a cause of action against Dr. Andershock and failed to produce any expert testimony regarding the proper standard of care that Respondent Hospital must follow in supervising its physicians and employees, the Hospital was also entitled to summary judgment with respect to Dulan's State law claims. (App. A at 15a-16a).

2. The Court of Appeals' Affirmance.

The two sentence per curiam opinion of the Court of Appeals is not published. The Court of Appeals entered its judgment on September 19, 2006. The Court of Appeals denied Petitioner's timely filed petition for rehearing and rehearing en banc on November 7, 2006.

REASONS FOR GRANTING THE WRIT

I. THE DISTRICT COURT AND THE PANEL ERRED AS A MATTER OF LAW IN REFUSING TO GIVE COLLATERAL ESTOPPEL EFFECT TO THE UNAPPEALED FINDINGS OF THE CMS UNIT OF THE DEPARTMENT OF HEALTH AND HUMAN SERVICES WHICH DETERMINED THAT RESPONDENT HOSPITAL'S EMERGENCY ROOM DEPARTMENT ENGAGED IN DISPARATE TREATMENT OF APPELLANT DULAN, VIOLATED EMTALA, AND ENDANGERED THE PUBLIC

A. Collateral Estoppel

The Department of Health and Human Services' Centers for Medicare and Medicaid Services (CMS) performed a legally mandated investigation of Respondent Huntsville Hospital and determined that the Hospital had violated the Emergency Medical Treatment and Active Labor Act (EMTALA) by delaying a medical screening for Petitioner Dulan to inquire about Dulan's insurance coverage. Specifi-

cally, CMS determined, among other things, that out of twenty emergency room patients it sampled, Dulan was the only patient who failed to receive a medical screening prior to answering questions about insurance. In the language of CMS:

> "This Hospital's Emergency Department failed to provide (1 of 20 sampled patients) Patient Identifier # 4 with an appropriate medical screening prior to inquiring about her insurance coverage." (App. D at 21a).

Additionally, CMS' findings stated that a person identified as Nurse # 4 stated:

> "[that it looks like [Petitioner Dulan] was registered before (she received a medical screening)," (App. D at 25a).

There is no dispute that patient identifier #4 is appellant Dulan. CMS also stated that the violation was so serious as to make the Hospital's emergency room a danger to the public. (App. E at 33a). This constituted a per se violation of EMTALA, which courts have uniformly held to proscribe disparate treatment of emergency room patients. *See, e.g., Harry v. Marchant*, 237 F.3d 1315, 1319 (11th Cir. 2001).

In Astoria Federal Savings and Loan Assoc. v Salimino, 501 U.S. 104, 108 (1991), this Court held that absent an "evident" Congressional "purpose to the contrary," courts may take it as a "given" that the federal common law doctrine of collateral estoppel is "presumed" to apply to the unreviewed findings of state and federal administrative agencies. *See University of Tennessee v Elliott*, 478 U. S. 758, 790, 794-99 (1986). *See also United States v Utah Constr. & Mining Co.*, 384 U.S. 394, 422 (1966).

Respondent Hospital's contention that CMS' finding that it violated EMTALA were not entitled to collateral estoppel effect because of due process concerns does not have merit. In *Matthews v Eldridge*, 424 U.S. 319, 339-40 (1976), this Court

held that pre-termination procedures almost identical to the ones utilized by CMS in this case[1] are "elaborate," and comport with due process.[2] Moreover, it is well established that "an administrative decision commands preclusive effect . . . if it resulted from a procedure that seems an adequate substitute for judicial procedure." Wright, Miller & Cooper, Federal Practice and Procedure: Jurisdiction 2d Sec. 4475, p. 183.

Significantly, this Court has favorably cited to one Circuit Court decision that suggested that the CMS' pre-termination procedure may accord even greater due process protections than the post termination process. *See O'Bannon v Town Court Nursing Center*, 447 U.S. 773, 778-79 at n.6 (1980). In *O'Bannon*, 447 U.S. 773 (1980), this Court cited with approval the Third Circuit Court of Appeals' holding that CMS' pre-termination process afforded due process to medical providers, stating that:

> "[The health care provider's] property interests were sufficiently protected by informal pretermination procedures and by the opportunity for an administrative hearing and federal-court review after benefits had been terminated:
>
> As was true in *Eldridge* (citation omitted), the decision not to renew a provider agreement is an easily documented, sharply focused decision in which issues of credibility and veracity play little role. It is based in most cases upon routine, standard, unbiased reports by health care professionals that were developed in the administrative rule-making process. Written submissions are adequate to allow the provider to present his case.

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[1] *See Northlake Community Hospital v. Department of Health and Human Services*, 654 F.2d 1234, 1241-42 (7th Cir. 1981) (finding the pre-termination procedures to be almost identical).

[2] *Matthews v. Eldridge*, 424 U.S. 319, 339-40 (1976), involved pre termination procedures utilized by the government prior to terminating individuals from receiving social security disability benefits.

> Given the extensive documentation that the provider is able to submit in response tote findings of the survey teams, the provider is unlikely to need an evidentiary hearing in order to present his position more effectively. In any event, there is ample opportunity to expand orally upon written submissions during the exit interview or in discussions during the survey itself. There is opportunity to submit additional evidence after notice of deficiencies is given, and the evidence upon which the recommendation of the survey team is based is disclosed fully to the provider. Moreover, the criteria used to evaluate the provider are well known in advance to the provider, and compliance is readily proved or disproved by written submission." (Emphasis added)

O'Bannon at 778-79. *See also, Northlake Community Hospital v The Department of Health and Human Services*, 654 F.2d 1234, 1241-42 (7th Cir. 1981).

Federal law empowers CMS to make "final enforcement decisions." *See* Statutes and Regulations portion of Petition. Therefore, CMS' findings in this case constituted a final, unappealed, and uncontested administrative decision that was entitled to collateral estoppel effect under well established Supreme Court precedent. *See Astoria Federal Savings and Loan Assoc. v. Salimino*, 501 U.S. 104, 108 (1991); *University of Tennessee v Elliott*, 478 U.S. 788, 790, 794-99 (1986); and *United States v Utah Construction & Mining Co.*, 384 U. S. 394, 422 (1966).

The Hospital chose not to put forth a defense to CMS' charges as it was entitled to do. Instead, the Hospital accepted CMS' findings of deficiencies and corrected those deficiencies. Accordingly, the undisputed findings of CMS became final without the necessity of a termination letter being issued.

Additionally, there was clearly an identity of issues for purposes of applying collateral estoppel since the findings by CMS related solely to the facts in Appellant Dulan's case. *See*

generally Montana v. United States, 440 U.S. 147, 153 (1979). Accordingly, the decisions of the lower court and the Circuit Panel in this case were erroneous as a matter of law.

B. Erroneous Application of Supreme Court Summary Judgment Standards

The court's role at the summary judgment stage is not to weigh the evidence or to determine the truth of the matter but, rather, to determine only whether a genuine issue exists for trial. *Anderson v. Liberty Lobby, Inc.*, 477 U. S. 242, 249 (1986). In doing so, the court must view the evidence in the light most favorable to the non-moving party and draw all reasonable inferences in that party's favor. *Matushita Elec. Inds Co. v Zenith Radio Corp.*, 475 U. S. 574, 587 (1986). Clearly, neither the District Court nor the Circuit Panel complied with this standard.

II. THE LOWER COURTS ERRED IN NOT FOLLOWING THE DOCTRINE *OF ERIE V. TOMPKINS* BY NOT FOLLOWING WELL ESTABLISHED ALABAMA LAW HOLDING THAT EXPERT TESTIMONY IS NOT REQUIRED WHERE PETITIONER INTRODUCES A RECOGNIZED STANDARD OR AUTHORITATIVE MEDICAL TEXT OR TREATISE TO PROVE WHAT IS OR IS NOT PROPER PRACTICE AS PETITIONER DID IN THIS CASE AND IN OTHER RESPECTS; NOR DID EITHER COURT GIVE A REASON FOR NOT FOLLOWING ALABAMA LAW

A. It Is Not Necessary For Petitioner To Produce An Expert Of Her Own In This Case

Alabama law does not require Petitioner to have an expert in this case. Well established Alabama legal precedent provides that Petitioner need not produce an expert witness where Petitioner introduces a recognized standard or authori-

tative medical text or treatise to prove what is or is not proper practice. *See Rosemont, Inc. v Marshall*, 481 So. 2d 1125, 1130 (Ala. 1985); *Bell v Hart*, 516 So.2d 562, 555 (Ala. 1987); *Holt v. Godsil*, 191, 193-94 (Ala. 1984); *Zills v. Brown*, 382 So. 2d 528 (Ala. 1980). Petitioner did that in this case by filing and introducing into evidence the ACC/AHA standards in the District Court during the summary judgment proceedings. Both the District Court and the panel failed to adhere to the doctrine set forth in *Erie v. Tompkins*, 304 U.S. 64 (1938)[3], by ignoring Alabama Supreme Court precedent. Moreover, neither of the lower courts stated a reason for its position. The same *Erie v. Tompkins* concerns are also present in the court's refusal to address, recognize, or distinguish controlling Alabama law on equitable estoppel which required that Appellees be estopped from filing their surprise motion to strike Appellant Dulan's identification of expert after agreeing to come to Washington, D.C. to depose Dulan's expert. *See Waldrop v. State Ex Rel. Broughton*, 465 So.2d 402, 403-04 (Ala. Civ. App. 1984), *citing* Alabama Supreme Court cases. *See also General American Life Ins. Co. v. Amsouth Bank*, 100 F.3d 893 (11th Cir. 1996).

Even though the Court of Appeals did not find that the District Court's decision to strike Petitioner's expert was an abuse of discretion or error as a matter of Alabama law, Petitioner should still be entitled to prove her Alabama medical malpractice case by first establishing that the standard of care for a case such as Dulan's is governed by the ACC/AHA standards. Dr. Cooper, Dulan's expert, stated in his report that the ACC/AHA standards are the "universally accepted guidelines . . . for the initial evaluation and treatment of all patients presenting to the emergency department with chest pain."

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[3] Jurisdiction in this case is based on both diversity and federal question. *Erie*, and its progeny, require that federal courts apply the same state law that a state court in that jurisdiction would have applied m deciding issues of state law.

Interestingly, Appellees never once stated that this is not the case. Moreover, Respondent-Appellee Dr. Andershock referred to these standards in answering a question during his deposition. (App. H at 69a).

Second, Petitioner can establish, through direct examination of Respondent Dr. Andershock and Respondents'-Appellees' other experts, the relevant standard of care, the damage to Dulan's heart proximately caused by Appellees' negligence, and conclusively show that Respondents have already admitted that they violated those standards. Dr. Andershock's deposition testimony and other evidence of record reveal that Respondents breached no less than the five crucial procedures set forth in the ACC/AHA standards in handling Petitioner's case.[4]

Dulan contends that Respondents Dr. Andershock and the Hospital owed her the duty to use such reasonable care, skill, and diligence as physicians qualified as ER physicians in the same general neighborhood, and the same general line of practice ordinarily have and exercise in a similar case. The same general neighborhood means the national medical neighborhood of reasonably competent physicians in the same line of practice acting in the same or similar circumstances. *See, Ala. Medical Liability Act*, Ala. Code sec. 6-5-484(a). *See Lamont v. Brookwood Health Servs., Inc.*, 446 So.2d 1018 (Ala. 1983); *Bradford v. McGee*, 534 So. 2d 1076 (Ala. 1988). Accordingly, the lower courts violated the spirit of this Court's decision in *Erie Railroad Company v. Tompkins*, 304 U.S. 64 (1938), by refusing to cite or address the clear applicability of Alabama Supreme Court precedent to this case.

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[4] Petitioner Dulan sets forth these five crucial breaches in her statement in this Petition.

CONCLUSION

For the foregoing reasons, the Court should grant a writ of certiorari in this case.

Respectfully Submitted,

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APPENDIX

APPENDIX A

UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF ALABAMA

NORTHEAST DIVISION

CV-02-CO-02445-

JEANNETTE ROGERS DULAN,

Plaintiff,

vs.

HUNTSVILLE HOSPITAL ASSOC., INC., *et at.*,

Defendants.

ORDER

In accordance with the memorandum of opinion entered contemporaneously herewith: Defendants' Motion to strike (Doc. 100) is DENIED, Plaintiff's motion to strike (Doc. 104) is DENIED, and Defendants' motion for summary judgment (Doc. 80) is GRANTED in all respects. Therefore, this case is DISMISSED with prejudice. Costs are taxed to the plaintiff.

Done this 5th day of August 2005.

/s/_____

L. SCOTT COOGLER

UNITED STATES DISTRICT JUDGE

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF ALABAMA
NORTHEAST DIVISION
CV-02-CO-02445-NE'

JEANNETTE ROGERS DULAN,
Plaintiff,

vs.

HUNTSVILLE HOSPITAL ASSOC., INC., *et at.*,
Defendants.

MEMORANDUM OF OPINION

I. Introduction.

The Court currently has for consideration a motion for summary judgment, which was filed by defendants Huntsville Hospital Association, Inc. ("the Hospital"), and Dr. Chris Andershock on December 28, 2004. (Doc. 80.) The plaintiff, Jeanette Dulan, has asserted claims against the Hospital under the Emergency Medical Treatment and Active Labor Act ("EMTALA"), 42 U.S.C. S 1395dd, et seq., for failure to provide an adequate medical screening examination and delaying treatment in order to inquire about the plaintiff's method of payment or insurance status, and under Alabama state law for vicarious liability, negligent supervision or maintenance, and negligent failure to terminate Dr. Andershock as an employee (Doc. 1.) Plaintiff has also sued Dr. Andershock for medical negligence and wantonness under Alabama state law, as well as discrimination based on race and/or sex under 42 U.S.C. § 1981. (Id.) The Court previously dismissed Plaintiff's EMTALA claim for failure to

provide medical treatment necessary to stabilize the, plaintiff. (Doc. 12.)

This Court also has for consideration Defendants' motion to .strike certain evidentiary materials filed by the plaintiff in opposition to summary judgment. (Doc. 100.) In her responsive submission to that motion, Plaintiff moved to strike several of Defendants' evidentiary exhibits. (Doc. 104.)

The issues raised in these motions have been briefed and are now ripe for decision. Upon due consideration, the motions to strike are due to be denied; Defendants' motion for summary judgment is due to be granted in all respects.

II. Facts.[1]

The plaintiff, Jeanette Dulan ("Dulan"), a fifty-five year-old African American female and resident of Maryland, developed chest pain on October 6, 2000, while visiting her parents in Huntsville, Alabama. (Doc. 103 at 1.) Dulan's mother took her to the emergency room at Huntsville Hospital,. And Plaintiff states that they arrived around 12:30 P.M. (Doc. 1 at Ex. A p.1.) Upon her arrival, an emergency. room employee met Plaintiff's car with a wheelchair and wheeled her into the hospital. (*Id.*) Plaintiff told the front desk clerk that she was experiencing chest pain and needed help. (*Id.*) The desk clerk asked her to complete a short form. She attempted to complete the form, but because she was in pain, she was unable to finish answering the questions. (*Id.*; Doc.

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[1] The facts set out below are gleaned from the parties' submissions of facts claimed to be undisputed, their respective responses to those submissions, and the Court's own examination of the evidentiary record. All reasonable doubts about the facts have been resolved in favor of the nonmoving party. See Info. Sys. & Networks Corp. v. City of .Atlanta, 281 F.3d 1220, 1224 (11th Cir. 20D2). These are the "facts" for summary judgment purposes only. They may not be the actual facts. See Cox v. Adm'r U.S. Steel & Carnegie Pension Fund, 17 F. 3d 1386, 1400 (11th Cir. 1994).

97 at Ex. 7 pp. 81, 86.) Plaintiff only recalls .providing her name and address. (Doc. 97 at Ex. 7 p. 86.) Plaintiff's mother then wheeled her into the waiting room. (Doc. 1 at Ex. A.)

Dulan waited approximately fifteen to twenty minutes before she was called to the triage area. (Doc. 97. at Ex. 7 pp. 92-93.) The triage nurse assessed Dulan's condition, then told her to return to the waiting area until someone called for her. (Doc. 103 at 4.) After waiting for *"not more* than fifteen minutes," an employee called Plaintiff to the registration desk. (Docs. 103 at 4; 97 at. Ex.7 p. 105.) The employee asked for personal information, such as Plaintiff's work, address, and insurance information. (Doc. 97 at Ex. 7 p. 103 Dulan did not have her insurance card, but she did tell the employee the name of her insurance company. (*Id.* at Ex. 7 p. 103.) She spoke with the registration clerk for less than five minutes (*Id.* at Ex. 7 p. 107), and then a nurse took her to the treatment area (Doc. 103 at 4). There, tests were run, X-rays were taken, and she was examined by Dr. Chris Andershock—a white male and the hospital's emergency room physician. (Doc. 1 at Ex. A p. 2.)

Later, a physician's assistant examined Plaintiff and she was given a nitro tablet. (*Id.*) Around 8:00 P.M. or 8:30 P.M., Plaintiff was examined by a cardiologist. (*Id.*) Dulan was admitted to the Hospital's cardiac care unit that evening. The cardiologist ordered a cardiac catheterization, and the procedure was performed that night. The catheterization showed that, Plaintiff was suffering an anterior myocardial infarction. (Doc. 103 at 4.) The Hospital proceeded to treat the heart condition, and Plaintiff underwent an angiography and angioplasty resulting in an intracoronary stent placement. (Doc. 1 at Ex. A p. 3.) Plaintiff was discharged on October 11, 2000, and commenced this action against the defendants on October 4, 2002.

III. Motions to Strike.

Defendants moved to strike certain evidentiary materials filed by the plaintiff in opposition to their motion for summary judgment, as well as certain statements made in Plaintiff's brief referring to that evidence. (Doc. 100.) Defendants argue that several of Plaintiff's evidentiary submissions are unsworn, hearsay, irrelevant, and/or inadmissible. (*Id.*) In her response submission, Plaintiff moved to strike some of Defendants' exhibits on the same grounds "if the court is inclined to agree with. Defendant[s]" that Plaintiff's evidence should be stricken. (Doc. 104 at 5.)

The materiality, relevancy, and potential admissibility of evidence at trial was carefully weighed by the Court during the summary Judgment process. Immaterial evidence, irrelevant evidence, and evidence that could not be reduced to admissible form, *see Macuba v. Deboer,* 193 F.3d 1316, 1322-23 (11th Cir. 1999), was rejected by this Court when it made its final decision on Defendants' summary judgment motion. For these reasons, the motions to strike will be denied.

IV. Summary Judgment.

Summary judgment is proper "if the pleadings, depositions, answers to interrogatories, and admissions on file, together with the affidavits, if any, show that there is no genuine issue as to any material fact and that the moving party is entitled to a judgment as a matter of law." Fed. R. Civ. P. 56(c). The party moving for summary judgment "always bears the initial responsibility of informing the district court of the basis for its motion, and identifying those portions of (the evidence) which it believes demonstrate the absence of a genuine issue of material fact." *Celotex Corp. v. Catrett,* 477 U.S. 317, 323 (1986). The movant can meet this burden by presenting evidence showing that there is no genuine dispute of material fact, or by showing that the nonmoving party has failed to

present evidence in support of some element of its case on which it bears the ultimate burden of proof. *Celotex, 477 U.S. at* 322-23. In evaluating the arguments of the movant, the court must view the evidence in the tight most favorable to the nonmovingparty. *Mize v. Jefferson City Bd. of Educ., 93* F.3d 739, 742 (11th Cir. 1996)

Once the moving party has met his burden, Rule 56(e) "requires the nonmoving party to go beyond the pleadings and by [his] own affidavits, or by the 'depositions, answers to interrogatories, and admissions on file,' designate specific facts showing that there is a genuine issue for trial.'" *Celotex,* 477 U.S. at 324 (quoting Fed. R. Civ. P. 56(e)). "A factual dispute is genuine only if a 'reasonable jury could return a verdict for the nonmoving party." *Info. Sys & Networks Corp. v. City of Atlanta* 281 F.3d 1220 1224 (11 th Cir.-2002) (quoting *United* States *v. Four Parcels of Real Property, 941* F. F.2d 1428, 1437 (11th Cir. 1991)).

A. EMTALA.

Plaintiff alleges that the Hospital is liable under EMTALA because it failed to provide her with an appropriate medical screening to determine whether an emergency medical condition existed in violation of 42 U.S.C. § 1395dd(a), and improperly delayed an appropriate medical screening and treatment in order to inquire about her insurance status in violation of 42 U.S.C. 5 1395dd(h). (Doc. 1.) The Hospital argues that Plaintiff does not have evidence sufficient to maintain her EMTALA claims and summary judgment is due to be granted. (Doc. 81.)

1. § 1395dd(a).

"In 1986, Congress enacted EMTALA in response to widely publicized reports of emergency care providers transferring indigent patients from one hospital to the next while the patients' emergency medical conditions worsened." *Harry v. Marchant,* 291 F.3d 767, 770 (11th Cir. 2002)

(en banc). "EMTALA was designed specifically to address this important societal concern; it was not intended to be a federal malpractice statute." *Id.*

"Section 1395dd(a) of EMTALA requires hospitals to provide persons requiring emergency medical treatment with 'an appropriate medical screening examination." ' *Holcomb v. Monahan,* 30 F.3d 116, 117 (11th Cir. 1994). "The Act does not define 'appropriate medical screening.' But, the congressional purpose behind the enactment of EMTALA supports the conclusion that this language only requires a hospital to provide. . . . patients with a medical screening similar to one which they would provide any other patient." *Id.* "[A] hospital has every right to tailor its screening procedures to a patient to account for her condition, stated. symptoms, and the determinations made by her persona[physician." *Nolen v. Boca Raton Cmty. Hosp., Inc.*, 373 F.3d 1151, 1155 (11th Cir. 2004).

Plaintiff contends that she has provided sufficient evidence that she did not receive an appropriate medical screening through the following:

(1) evidence that the Hospital failed to follow "clear, universally-accepted guidelines" and/or its own procedures (*Id.* at 3, 7, 18, 23, 2-4, 62-66, 70);

(2) evidence that there are racial disparities in the Hospital's "administration of aspirin to persons visiting the Hospital's emergency room" (*Id.* at 8, 18, 25);

(3) studies showing that there is nation-wide 'discrimination on the basis of race and. gender in treating victims of heart attack (*Id.* at 51, 55, 70-71, 74-75);

(4) (4) evidence that Dr. Andershock reported Plaintiff's pain as "mild" when she was suffering "visible" excruciating pain (*Id.* at 53, 58, 68);

(5) the. fact that the Hospital did not diagnose Plaintiff's heart condition for approximately eight hours (*Id.* at 53, 55); and

(6) evidence that before Plaintiff's heart condition was diagnosed, it was the Hospital's intent to discharge Plaintiff that day and have her return three days later for testing (*Id.* at 53, 56, 59). Plaintiff contends that this evidence demonstrates that' the Hospital "never had any intention of providing [Plaintiff with] 'a medical screening calculated to identify critical medical conditions.'" (*Id.* at 57.)[2]

However, "EMTALA was not intended to establish guidelines for patient care, to replace available state remedies, or to provide a federal remedy for medical negligence." *Harry v. Morchant,* 291 F.3d 767, 773 (11th Cir. 2002). A contention that a hospital did not follow certain policies or guidelines for victims exhibiting symptoms of heart attack is not sufficient to prevail on an EMTALA *claim. Nolen v. Boca Raton Cmty. Hosp.,* 373 F.3d 1151, 1154-55 (11th Cir. 2004). Evidence of widespread racial or gender-related disparities in the healthcare provided to individuals exhibiting symptoms of heart attack does not show that Plaintiff experienced a disparate medical screening in this case. In addition, even assuming that Plaintiff's study showing that African-American patients with heart problems were treated

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[2] The Court notes that Plaintiff consistently cites to her Complaint throughout her response brief in opposition to summary judgment when referring to "evidence" in her favor. At summary judgment, Plaintiff is required to go beyond the pleadings to show that she has evidence to establish a genuine issue of *fact* for trial. *Celotex Corp. v. Catrett,* 477 U.S. 317, 324 (1986).

differently at the Hospital with regard to the administration of aspirin was statistically significant, the study relates to the treatment of diagnosed heart conditions and not the screening examination at issue in this case.[3] (Doc. 96 at Ex. 3 p. 100, Ex. 4 p. 101.)

"As long as the Hospital screened [Plaintiff] in a manner consistent with the screening that any other patient in the care of a private physician would have received, there can be no liability under the EMTALA." *Nolen v. Boca Raton Cmty. Hosp.*, 373 F.3d 1151, 1154-55 (11th Cir. 2004). The Hospital in this case has presented evidence that Plaintiff's screening was appropriate, and consistent with the quality of screening that a similarly-situated patient would have received. Plaintiff has "failed to present even .a single affidavit of *a physician* to dispute the adequacy of the screening provided by the Hospital." *Id.* at 1155 (emphasis added).[4]

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[3] The study also shows that Plaintiff received aspirin. (Doc. 82 at Ex. L Attach. C.)

[4] Plaintiff contends that she has submitted the transcript of a radio broadcast where a "Dr. Eaton," who allegedly stated that it was his opinion that the Hospital had breached its duty of care to Plaintiff and violated EMTALA. This evidence was apparently attached to a brief in opposition to Defendants' motion to dismiss. (Doc. 95 at 11.) However, the presiding Judge in this case when the motion to dismiss was filed did not require that briefs in support or opposition be made a part of the record, and the brief and evidence referenced by Plaintiff was not filed and is not a part of the record. Therefore, that evidence is not before the Court for consideration at this time.

Plaintiff also emphasizes the findings of the Centers for Medicare & Medicaid Services (CMS) throughout her response brief. Plaintiff has attempted to argue that CMS's legal conclusions are binding on this Court. The Court rejected that assertion when it denied Plaintiff's motion to have the findings receive collateral estoppel effect. (Doc. 87.) CMS's legal conclusions are also not evidence that Defendants are liable. (The Court notes, however, that CMS's legal conclusions only pertain to an

Plaintiff's contentions that Dr. Andershock mischaracterized her "visible" pain and that the Hospital took too long to discover her heart condition and intended to discharge her before the heart condition was accurately diagnosed are also not sufficient to prevail on her EMTALA claim. The Eleventh Circuit Court of Appeals has soundly rejected the "argument that the 'appropriateness' of the screening should be determined by its adequacy in identifying the patient's illness." *Holcomb v. Monahan*, 30 F.3d 116, 117 (11th Cir. 1994). "Section 1395dd(a) is not designed to redress a negligent diagnosis by the hospital." *Id.* "It is not intended to ensure each. emergency room patient a correct diagnosis." *Harry v. Marchant*, 237 F.3d 1315, 1319 (11th Cir. 2001), rehearing en banc granted on other grounds and opinion vacated by 259 F.3d 1310 (11th Cir. 2001), reinstated in part on rehearing by 291 F.3d 767 (11th Cir. 2002).[5] Although the Hospital did not immediately discover Plaintiff's heart condition, the condition was ultimately diagnosed, and Plaintiff was admitted and treated for the condition. Plaintiff has not produced any competent evidence to support her claim that the Hospital violated 5 1395dd(a) by failing to provide her with a medical screening similar to one that they would provide any other patient.

2. S 1395dd(h).

Section 1395dd(h) of EMTALA provides that hospitals "may not delay provision of an appropriate medical screening examination . . . or further medical examination and treatment

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alleged violation of §1395dd(h), and not § 1395dd(a).) Even if the Court accepts CMS's findings that the Hospital violated certain alleged policies and procedures, this is not sufficient to establish a violation of § 1395dd(a). *Nolen v. Boca Raton Cmty. Hosp.*, 373 F.3d 1151, 1154-55 (11th Cir. 2004).

[5] Rehearing en banc was granted solely to determine the scope of EMTALA's stabilization requirement." *Harry v. Marchant*, 291 F.3d 767, 769 (11th Cir. 2002). This requirement is not at issue in this case.

in order to inquire about the individual's method of payment or insurance status." 42 U.S.C. S 1395dd(h). Plaintiff alleges that the Hospital violated this provision when it asked her for insurance information. Before screening, Plaintiff was asked to fill out a short registration form, which Plaintiff did not complete.. Plaintiff then waited about fifteen to twenty minutes before being taken to the triage area. After being assessed in triage, 'Plaintiff was returned to the waiting room. Approximately fifteen minutes later, a hospital employee inquired about information Plaintiff had not provided on the registration form, including her insurance information. Plaintiff did not have her insurance card, but she provided the name of-the insurance company. After speaking with this hospital employee for no more than five minutes, a nurse returned and took Plaintiff to the treatment area.

The fact that the Hospital inquired about Plaintiff's insurance information does not establish a violation of § 1395dd(h). Rather, Plaintiff must show- that her medical screening or treatment was delayed because of the inquiry. Federal Regulations provide. that "[h]hospitals may follow reasonable registration processes for individuals for whom examination or treatment is required by this section, including asking whether an individual is insured and, if so, what that insurance is, as long as that inquiry does not delay screening or treatment." 42 C.F.R. S 489.24(d)(4).(iv); *see also Jakubiec v. Sacred Heart Health Sys:, Inc.*, 2005 WL 1261443 at *3 (W.D. Fla. May 27, 2005) ("Requiring- an individual to sign a standard intake form does not violate EMTALA, and hospitals may follow reasonable registration processes, including asking whether the individual is insured.") (quoting *Quinn v. BJC Health Sys.*, 364 F. Supp. 2d 1046 (E.D. Mo. 2005)). Plaintiff has not produced any

evidence that her screening or treatment was delayed on account of the Hospital's inquiries into her insurance status.[6]

Even if this Court concluded that Plaintiff did have some evidence that the Hospital violated 5 1395dd(h), Defendant argues that EMTALA only allows a plaintiff *to recover damages for* personal harm suffered "as a direct result" of the . violation and appropriate equitable relief. 42 U.S.C. S 1395dd(d)(2)(A). (Doc. 81 at 43.) Plaintiff has not addressed the damages issue in her brief, and has not presented any competent medical evidence that she suffered any personal harm as a result of the alleged delay caused by the Hospital's inquiries into her insurance status.

For these reasons, the *Hospital's motion* for summary judgment on Plaintiff's EMTALA claims is due to be granted.

B. Section 1981.

Plaintiff contends that Dr.. Andershock violated 42 U.S.C. 1981 by denying her the same "'benefits, privileges, terms, and conditions of the contractual relationship,' that [he]

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[6] Plaintiff argues that CMS's conclusion that the Hospital "failed to avoid delay in providing a medical screening exam in order to inquire about the individual's method of payment or insurance status," creates an issue of material fact in her favor. (Doc. 96 at Ex. 1.) However, legal conclusions are not evidence or helpful in this instance. CMS concluded that the Hospital committed a violation because "it was determined . . . [that the] Hospital's Emergency Department staff failed to provide [Plaintiff] with an appropriate medical screening prior to inquiring about her insurance coverage." (Doc. 96 at Ex. 2.) As discussed by this Court, this is not the appropriate legal standard for assessing a violation of § 1395dd(h) of EMTALA.. A defendant does not violate. the provision simply by asking about insurance before the medical screening. Rather, a plaintiff must establish that the defendant delayed the screening or treatment because of the inquiry. According to Plaintiff, when the nurse returned, she wheeled Plaintiff directly to the treatment area and ignored the Hospital employee's protests that she did not have all of Plaintiff's registration information.

would have afforded to a similarly. situated White/Caucasian patient. (Doc. 1 at 20-21.) Dr. Andershock argues that he is entitled to summary judgment on Plaintiff's §1981 claim because:

1 Plaintiff cannot establish the existence of an underlying contract to support her claim;

2 (2) a §1981 claim is unavailable against state actors such as Dr. Andershock; and

3 (3) Plaintiff has not presented evidence of intentional discrimination. (Doc. 81 at 43.)

Section 1981 provides that "[a]ll persons-within the jurisdiction of the United States shall have the same right in every State and Territory to make and enforce contracts, to sue, be parties, give evidence, and to the full and equal benefit of all laws and proceedings for the security of persons and property as is enjoyed by white citizens." 42 U.S.C. 9 1981: A plaintiff alleging a non-employment based discrimination claim under 9 1981 must establish three things in order to survive a motion for summary judgment: "(1) he or she is a member of a racial minority; (2) the defendant had an intent to discriminate on the basis of race; and (3) the discrimination concerned one or more of the activities enumerated in the statute (in this case, the making and enforcing of a contract)." *Rutstein v. Avis Rent-A-Car* Sys., Inc., 211 F.3d 1228, 1235 (11th Cir. 2000).

Plaintiff does not address the issue of contractual relationships in her responsive submission to the Court. In fact, there is no evidence in the record of an express contract with Dr. Andershock or the Hospital; there is no evidence of an attempted contract with Dr. Andershock or the Hospital; and Alabama law does not recognize implied contracts to exercise due care in the delivery of medical services by a physician or a hospital. *Marsh v. St. Margaret's* Hospital., 535 So. 2d 147, 149-50 (Ala. 1985); Lemmond *v .Sewell, 473*

So. 2d 1047, 148-49 (Ala. 1985); Berry v. Druid City Hospital Board, 333 So. 2d 796, 799 (Ala. 1976); *Smith v. Housten County Hospital Bibard,* 255 So. 2d 328, 330 (Ala. 1971). Because Plaintiff has failed to establish the existence of a contractual relationship to exercise due care in her medical treatment sufficient to support her claim under § 1981, Dr. Andershock is entitled to summary judgment on this issue.

Even if there was a contractual relationship, Plaintiff's § 1981 claim fails because she has not produced any evidence that "the defendant had an intent to treat . . . her less favorably because of [her race]." Rutstein, 211 F.3d at 1235. Section 1981 claims require proof of intentional discrimination. *General Bldg. Contractors* Assn, *Inc. v. Pennsylvania,* 458 U.S. 375, 391, 102 S. Ct. 3141, 3150, 73 L. Ed. 2d 835 (1982) (holding that "§ 1981, like the Equal Protection Clause, can be violated only by purposeful discrimination"); *Ferrill v. The Parker Group, Inc.,* 168 F.3d 468, 472 (11th *Cir. 1999); Donaire v. NME Hosp.,* 27 F.3d 507, 509 (11th Cir. 1994) *Brown v. Am. Honda MotorCo.,* 939 F.2d 946 (11th Cir. 1991); *Smith v. Popp* Clinic, P.A., 808 F.2d 1449, 1453 (11th Cir. 1987); *Freeman v. Motor Convoy, Inc.,* 700 F.2d 1339, 1350 (11th Cir. 1983). Plaintiff offers the following evidence *in support of her claim: (1)* the fact that she is African American and Dr. Andershock is White/Caucasian, (2) articles alleging that there is widespread racial discrimination in healthcare, *particularly in the* treatment of heart disease, and (3) Plaintiff's disparity study that allegedly shows a difference in the administration of aspirin to African Americans versus Caucasians in the Hospital's emergency room. The latter two *items are* not at all relevant to Dr. Andershock's individual treatment of Plaintiff on October 6, 2005, and the fact that -Defendant is White/Caucasian is simply not sufficient to establish that he had an intent to discriminate against Plaintiff because she is

African American. Therefore, summary judgment is due to be granted on Plaintiff's § 1981 claim.

C. State Law Medical Malpractice.

Plaintiff alleges Alabama. state taw medical malpractice claims against both the Hospital and Dr. Andershock. Plaintiff maintains that Dr.Andershock is liable for medical negligence and wantonness, and that the Hospital is (1) vicariously liable for Dr. Andershock's negligence, and (2) negligent in "failing to have in place and/or failing to correctly utilize proper procedures and protocols for the evaluation of heart attack patients in its emergency room (such as [P]laintiff) and negligently granting, maintaining, and failing to terminate defendant Dr. Andershock." (Doc. 1 at 19.) Defendants contend that they are entitled to summary judgment on all of Plaintiff's state taw claims.

Under Alabama law, in order to prove liability on her medical malpractice claims against the Hospital and Dr. Andershock, Plaintiff must establish that a duty of care exists, the appropriate standard of care, the defendant(s) breached that standard of care, and such breach was the proximate cause of injury to the plaintiff. *Bradley v.* Miller, 878 So. 2d 262, *266 (Ala. 2003); Hauseman v. Univ. of Alabama Health Servs., 793 So.* 2d 730, 734 (Ala. 2000); *Looney v.* Davis, 721 So. 2d 152, 157 (Ala. 1998). In order to survive a motion for summary judgment, generally a plaintiff must "submit competent expert medical testimony to prove that the defendant violated the standard of care [established by law]." *Wilson v. Teng*, 786 So. 2d 485, *498 (Ala. 2000); see also, e.g., Schuffert v. Morgan,* 777 So. 2d 87, 91 (Ala. 2000); *Looney v.* Davis, 721 So. 2d 152, 157 (Ala. 1998); *Univ. of Alabama Health Servs. Found., P.C. v. Bush,* 638 So. 2d 794, 798 (Ala. 1994). Defendants have provided evidence that the standard of care was *satisfied* in this case. Plaintiff has failed to produce any expert testimony that refutes this evidence.7 Therefore, Defendants are entitled to summary judgment on

Plaintiff's medical malpractice claims of negligence, wantonness, and vicarious liability.

Because Plaintiff has not established that she has a cause of action against Dr. Andershock for negligence,-and- because Plaintiff has also failed 'Plaintiff has also failed to produce any expert testimony regarding the applicable standard of care and whether Defendants' alleged breach proximately caused Plaintiff's alleged injury to produce any expert testimony regarding the proper standard of care that a Hospital must follow in supervising its physicians and employees, or the alleged breach of that standard, *see Mann v. Health Care Auth. of the City of Huntsville*, 653 So. 2d 941, 942 (Ala. 1995), the Hospital is entitled to summary judgment on Plaintiff's claim for negligent supervision and maintenance of Dr. Andershock.

V. Conclusion.

For the reasons stated above, the motions to strike filed by both Defendants and Plaintiff will be denied; however, Defendants' motion for summary judgment is due to be granted in all respects. A separate order in conformity with this opinion will be entered.

Done this 5th -day of Augusts 2005.

/s/_____
L. SCOTT COOGLER
UNITED STATES DISTRICT JUDGE

APPENDIX B

IN THE UNITED STATES COURT OF APPEALS
FOR THE ELEVENTH CIRCUIT

[Filed Sept. 19, 2006]

D.C. Docket No. 02-02445-CV-LSC-NE

No. 05-14799

JEANNETTE ROGERS DULAN,
Plaintiff-Appellant Cross-Appellee,

v.

HUNTSVILLE HOSPITAL ASSOCIATION, INC., ANDERSHOCK, DR.; M.D.; Emergency Room Physician of dft Huntsville Hospital,
Defendants-Appellees Cross-Appellant.

Appeals from the United States District Court
for the Northern District of Alabama

Before BIRCH, PRYOR and FAY, Circuit Judges.

PER CURIAM:

After conducting oral argument, thoroughly reviewing the briefs of the parties and the record in this case, we discern no error on the part of the district court in awarding judgment to the defendants in this case. Accordingly, the judgment of the district court is AFFIRMED.

IN THE UNITED STATES COURT OF APPEALS

FOR THE ELEVENTH CIRCUIT

[Filed Sept. 19, 2006]

D.C. Docket No. 02-02445-CV-LSC-NE

No. 05-14799

JEANNETTE ROGERS DULAN,

Plaintiff-Appellant Cross-Appellee,

v.

HUNTSVILLE HOSPITAL ASSOCIATION, INC., ANDERSHOCK, DR.; M.D.; Emergency Room Physician of dft Huntsville Hospital,

Defendants-Appellees Cross-Appellant.

Appeals from the United States District Court for the Northern District of Alabama

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JUDGMENT

It is hereby ordered, adjudged, and decreed that the attached opinion included herein by reference, is entered as the judgment of this Court.

Entered: September 19, 2006

For the court: Thomas K. Kahn, Clerk

By: Gilman, Nancy

APPENDIX C

IN THE UNITED STATES COURT OF APPEALS
FOR THE ELEVENTH CIRCUIT

[Filed Nov. 7, 2006]

No. 05-14799-FF

JEANNETTE ROGERS DULAN,

Plaintiff-Appellant Cross-Appellee,

v.

Huntsville Hospital Association, INC.,
ANDERSHOCK, DR.; M.D.; Emergency Room
Physician of dft Huntsville Hospital,

Defendants-Appellees Cross-Appellants.

On Appeal from the United States District Court for the
Northern District of Alabama

ON PETITIONS) FOR REHEARING AND PETITIONS) FOR REHEARING EN BANC

Before: BIRCH, PRYOR and FAY, Circuit Judges.

PER CURIAM:

The Petition(s) for Rehearing are DENIED and no Judge in regular active service on the Court having requested that the Court be polled on rehearing en banc (Rule 35, Federal Rules of Appellate Procedure), the Petition(s) for Rehearing En Banc are DENIED.

ENTERED FOR THE COURT:
UNITED STATES CIRCUIT JUDGE

APPENDIX D

Continued From page 2

1:10 p.m., when asked if the Emergency Department was busy when she presented to the Emergency Department on 10/6/00, PI#4 stated "I couldn't tell-that it was very busy. When I came in there was someone sitting there. I was the only one in crisis."

Review of the Emergency Room Log dated 10/6/00 revealed between 12:07 p.m., and 1:08 p.m., twenty two (22) patients presented to this facility's Emergency Department Sixteen (16) of the twenty two (22) were discharged home/self care. One (1) patient eloped from the Emergency Department and five (5) patients were admitted. At 12:16. p.m., a patient was admitted with Acute Myocardial Infarction. At 12:38 p.m., a patient Was admitted with Acute Polyarthritis. At 12:46 p.m., a patient was admitted with Fracture of Lumbar 2. At 12:51 p.m., a patient was admitted with VP Revision.

Further review of the Emergency Department Record for PI#4 dated 10/6/00 revealed laboratory test Prothrombin Time and Partial Prothrombin Time were not ordered in the Emergency Department for PI#4. The Emergency Department medical record lacks evidence Emergency Department Nursing staff consulted with the Emergency Department Physician in an attempt to determine if the Prothrombin Time and Partial Prothrombin Time laboratory studies were needed.

Continued review of the Emergency Department Record for Patient Identifier (PI) #4 revealed the initial triage information was documented on 10/6/00 at 1:02 p.m. On 10/6/00 at 2:40 p.m. PI#4 was seen by the Emergency Room Physician, Prior to 2:40 p.m., PI#4's Emergency Room medical record lacks evidence the Emergency

Continued From page 3

Room Physician was notified of PI#4's complaint of chest pain.

On 6/27/03 at 8:00 a.m. an interview was conducted with Nurse #1. When asked if she reported her findings to the Emergency Room Physician Nurse #1 stated. . . . "I must have not spoken to the doctor. I documented I reported to another nurse.

Refer to A408

489.24(c)(3) DELAY IN EXAMINATION OR TREATMENT

A participating hospital may not delay providing an appropriate medical screening examination required under paragraph (a) of this section or I further medical examination and treatment required under paragraph (c) in order to inquire about the individual's method of payment or insurance status.

This STANDARD is not met as evidenced by:

Surveyor: 01708

During the investigation of Complaint #AL00000609 it was determined through interviews, review of Patient Identifier (PI) #4's Emergency Department Medical Record, review of this facility's Emergency Department Orientation Manual, and review of this facility's policies and procedures this Hospital's Emergency Department staff failed to provide (1 of 20 sampled patients) Patient Identifier #4 with an appropriate medical screening prior to inquiring about her insurance coverage. Patient Identifier (PI)#4 presented t6the Emergency Department of this facility on October 6, 2000 with

Continued From page 4

a medical emergency, cardiac in nature. PM presented with complaints of "intense chest pressure, nausea, and vomiting. P#4 was

later diagnosed with an Anteroapical infarct requiring emergency heart catheterization and stent placement PI#4 was directed to registration where an attempt was made to obtain insurance information prior to PI#4 receiving a medical screening. This was apparent by the following:

An interview was conducted with Patient Identifier #4 on 6/26/03 at 1:10 p.m. Patient Identifier #4 stated" I arrived at the Emergency Department (on 10/6/00) at 12:30 p.m. . . . I was asked for Insurance at 1:15 p.m. to 1:30 p.m. I was in the waiting room in a state of heart attack, weak withintense pressure in my chest. When I could not sit up I was leaning out of the wheelchair trying to get in position (in a comfortable position). The nurse acted like I had stubbed my toe. The nurse told me to go back out and wait for registration at a window; Counter 3. The Counter 3 lady (the registration clerk) was not going to send me back because I didn't have the Insurance card. . . . . The triage nurse is the one who old me to go back and wait for registration. The other nurse (name unknown) saw that I was having chest pain. The other nurse said-we have to take her back right now. The registration person said you can not take her back I don't have the Insurance information."

Review of the Emergency Department Record for PI#4 dated 10/6/00 revealed a triage time of 1302 (1:02 p.m.). On 10/6/00 PI#4 presented with a complaint of chest pain since this a.m. (the morning of 10/6/00). Vital signs are documented as blood pressure 182/96, pulse 46, respirations 20, and body temperature 96.7 Oxygen saturation is documented as 100%.

Continued From page 5

An interview was conducted with Nurse #4 on 6/27/03 at 9:30 a.m. Nurse #4 stated the 1302 (1:02 p.m.) time is actually the time the person assigned to the front desk made his documentation.

During an interview with Nurse #1 on 6/27/03 at 8:33 a.m. this staff nurse explained that the person documenting the above information (triage time, chief complaint, vital signs, and oxygen saturation) was not the triage nurse but the attendant at the desk at the time PI#4 presented to this facility's Emergency Department. When asked what PI#4 was doing between 1302 (1:02 p.m.) and 1310 (1:10 p.m., the triage time) Nurse #1 stated "between those times she (PI#4) was filling out the half pass and the information was being put in the computer."

On 6/27/03 at approximately 10:40 a.m., an interview was conducted with Nurse #3. Nurse #3 stated she was the nurse who triaged PI#4 on 10/06/00. When asked if she entered the priority on PI#4's emergency room medical record Nurse #3 stated "no, the person at the front desk did." When asked what her findings were Nurse #3 stated "that she (PI#4) had chest pain for 45 minutes duration and nausea and vomiting." When asked she noted the Emergency Room Physician immediately of PI#4's complaint Nurse #3 stated "I would have called the charge nurse for a bed" When asked if PI#4 received a medical screen prior to going to registration Nurse #3 stated "by the triage nurse. That (the medical screen) would occur as soon as she could be placed in a room on a bed so the physician could assess her."

On 6/27/03 at 8:00 a.m. an interview was

Continued From page 6

conducted with Nurse #1. Nurse #1 stated she was the first nurse to see PI#4 (a time of 1:35 p.m., is documented reflecting when Nurse #1 assessed PI#4). When asked what (PI#4's) complaint was at the time she saw PI#4 on .10/6/00 Nurse #1 stated "general anterior chest pain radiating to neck and shoulder." When asked if she reported her findings to the Emergency Room Physician Nurse #1 stated "the standard is to document and get chart to rack for physician to see. If you have concerns or priority one patient you would go to the doctor.... I must have not spoken to the doctor. I documented I reported to another nurse." When asked what her

findings were based on the electrocardiogram Nurse #1 stated the findings were abnormal."but I didn't see anything regarding marked ischemia or injury." When asked if blood pressures of 182/92 and 204/80 were causes for concern Nurse #1 stated "they support the priority 2. She's a patient that need to be urgent."

An interview was conducted with Registration Clerk #1 on 6/26/03 1:40 p.m. When asked when she ask for insurance information from patients Registration Clerk #1 stated 'the patient comes to the front desk The nurse will ask them to fill out a half pass with their name, date of birth, and social security number. They put their complaint on the half pass. The half pass is put in the box. The triage nurse has the patient doing the blood pressure and getting a bed. The triage nurse will see the patient first. The person assigned to the half pass put the information in the computer to create a medical record. Then we (registration clerks) are paged to go to the bedside to complete the registration or the patient comes to the desk." When asked to explain her task Registration Clerk #`1 stated "make sure address is correct for billing, make sure demographics are

Continued From page 7

correct, obtain emergency contact information, employment information, obtain insurance -information, get contract signed, and go back to complete, put information in (the system). Registration Clerk #1 further stated "sometimes the doctor and the. nurse are already in there (in the treatment room with the patient) when we get the information." When asked how she verify Insurance Coverage Registration Clerk #1 stated "we call the 1 800 number on the back of the insurance card." When asked to tell this writer when the call to verify insurance would be made Registration Clerk #1 stated "during the registration process." When asked what indication writer would find on the medical record to reflect when insurance coverage was verged Registration Clerk #1 stated "we put the I insurance

information on the sheet and that let's the doctor know they have insurance" At 2:20 p.m., when asked to explain the time frames on the Emergency Record for PI#4, Registration Clerk #1 stated "at 13:10 (1:10 p.m.) she (PI #4) was triaged At 13:35 (1:35 p.m.)-she was put in a room and registration was paged. At 13:40 (1:40 p.m.) registration was completed. Then the Doctor took over."

An interview was conducted with Registration Clerk #2 on 6/26103 at 2:20 p.m. When Registration Clerk #2 was asked to walk this writer through the registration process she stated "the patient would come in and fill out a half pass. The half pass has vague demographic information, the reason for the visit and the address and telephone number. The Triage nurse will see the patient, assess the patient, and prioritize the patient. The Triage nurse gives the half pass to the first pass person who put you in the computer, get acknowledge agreement, and I makes a bracelet. The patient is completely

Continued From page 8

triaged (by the nurse) then we are paged to the room and we complete registration. We get the insurance information at the bedside. Unless the beds are full then the patient is called to a booth: We get the work history and guarantor's information and "stuff" (such as) insurance."

During the continued interview with Nurse #4 on 6/27/03 at 9:30 a.m., when this writer attempted to obtain specifics regarding the registration of PI#4 Nurse #4 stated "looks like she was registered before (she received a medical screening)." Nurse #4 also confirmed PI#4 came in to this facility's Emergency Department on 10/6/00 at 1302 (1:02 p.m., per documentation of person at front desk) and was seen by the Emergency Room Physician at 1440 (2:40 p.m.). I Nurse #4 also stated patients are registered after triage.

Continued review of the Emergency Department Record for PI#4 dated 10/6/00 revealed at 1:10 p.m. PI#4 complained of Chest pain tightness midstream worse with inspiration. Onset at forty five

minutes ago. Skin warm and dry, color within normal limits. Positive (+) for nausea and vomiting. PI#4 complained at 1:35 p.m., of general anterior chest pain to left neck and shoulder. This occurred while talking . PI#4's vital signs at 2:00 p.m., were documented as pulse 48, respirations 16, blood pressure 201/80, body temperature 100.1 rectally. PI#4 continued to complain of pain and vomited large amount of undigested food and liquid PI #4 was seen by Emergency Room Physician #1 at 2:40 p.m. At 7:15 p.m., PI#4 was transported to Cardiac Care Unit. At 7:20 p.m. PI#4 was seen by Physician #3. At 8:30 p.m., PI#4 was sent to the Cath Lab.

An interview was conducted on 6/26/03 at 5:15

Continued From page 9

p.m. with Physician #3. Physician #3 stated the trend is to get an electrocardiogram and initiate treatment with in a half hour.

During continued interview with Patient Identifier #4 on 6/26/03 at 1:10 p.m., this complainant stated "the Cardiologist diagnosed me in a state of heart attack. I went directly to the

Catheterization laboratory. A stent was put in and then I went to the Cardiac Care Unit."

Review of the Cardiac Catheterization Report dated 10/6/00 revealed it contains the following: "Indication: . . . "She (PI#4) has come to the Emergency Room but initially her symptoms were felt related to pericarditis but due to persistent pains we are now asked to come in for emergent cardiac catheterization.

. . .

Procedure In Detail: The patient was brought to the cardiac catheterization laboratory in a fasting state. Unfortunately by this time she was already nine hours after onset of symptoms

. . .

Angiography and left ventricular angiography were performed using standard Judkins catheters demonstrating total occlusion of the mid left anterior descending coronary

artery after a diagonal that bifurcated. This was the culprit site."

Review of Physician #2's Discharge Summary containing a discharge date of 10/11/00 for PI#4 revealed the following:

"1. Anteroapical infarct related to total occlusion of the mid left anterior descending coronary artery successful percutaneous transluminal coronary angioplasty and recannulization late in her course.

2. Moderate disease of diagonal and obtuse marginal.

3. Mildly depressed left ventricle function with

Department of Health & Human Services
Center for Medicare & Medicaid Services
61 Forsyth St. Suite 4T20
Atlanta, Georgia 30303-8909

[Logo]

M ... hh062003-res

August 26, 2004

Mr. L. Joe Austin, CEO Huntsville Hospital

101 Silvey Road

Huntsville AL 35801

RE: Complaint Control #062003

Dear Mr. Austin:

This is written in regard to my letter to you dated August 10, 2004 concerning the noncompliance of Huntsville Hospital, in Huntsville, Alabama Because of noncompliance, I had advised you that the hospital's Medicare certification would be terminated effective September 2, 2004. Subsequent to my letter, I received documentation from you that adequately addressed the cited deficiencies. The Alabama State Agency conducted a follow-up visit on August 25, 2004, and determined that action had been taken to correct the deficiencies that were cited, and that procedural changes had been made giving reasonable assurance that a similar violation will not recur.

Based on the documentation I have received from the August 25, 2004 State Agency follow-up survey, I am withdrawing the termination action as stated in the previous correspondence. However, we are required to refer the case to the Office of the Inspector General for their decision as to whether civil monetary penalties should be imposed

The cooperative attitude exhibited by you and your staff is greatly appreciated. Please contact my office if you have any questions and speak with Ann Pfeiffer Huffman at (404) 562-7462 or Colleen Sandmann or (404) 562-7458.

Sincerely,

Sandra M. Pace

Associate Regional Administrator
Division of Survey and Certification

Cc: Albama State Agency

SMP/aph

APPENDIX E

Department of Health & Human Services
Centers for Medicare & Medicaid Services
61 Forsyth St. Suite 4T20
Atlanta, Georgia 30303-8909

Reference: M: hh062003-193

September 14, 2004

Ms. Jeanette Rogers Dulan
16110 Parklawn Place
Bowie, MD 20716

Dear Ms. Dulan:

We have received the report from the investigation that we authorized in response to your allegation that Huntsville Hospital, in Huntsville, Alabama, violated the emergency care obligations of 42 CFR § 489.24, Responsibilities of Medicare Participating Hospitals, in Emergency Cases and/or the related provisions at 42 CFR § 489.20. The situation you described was found to be a violation of 42 CFR 489.24.

Enclosed is a copy of the letter we sent to the hospital. If the hospital violated 42 CFR §489.24, we are notifying the Office of the Inspector General because it has responsibility for the enforcement of the civil monetary penalties prescribed by §1867 of the Social Security Act (the Act). In addition, we are notifying the Office for Civil Rights because it may take action under the Hill-Burton Subpart G. Community service regulations. Also based on your individual situation, you may wish to consider the civil enforcement provisions of section 1867 of the Act on an independent basis.

It is our goal to help health care facilities provide the best possible care to patients. Thank you for bringing this matter to our attention.

Sincerely,

Sandra M. Pace
Acting Associate Regional Administrator
Division of Survey and Certification

Enclosure
SMP/aph

Department of Health & Human Services
Centers for Medicare & Medicaid Services
61 Forsyth St. Suite 4T20
Atlanta, Georgia 30303-8909

Refer to: hh062003-190

August 10, 2004

Mr. L. Joe Austin, CEO
Huntsville Hospital
101 Silvey Road
Huntsville, AL 35801

Dear Mr. Austin:

To participate in the Medicare Program, a hospital must meet the requirements established under Title XVIII of the Social Security Act (the Act) and the regulations established by the Secretary of Health and Human Services under the authority contained in §1861(e) of the Act. Further, § 1866(b) of the Act authorizes the Secretary to terminate the Provider agreement of a hospital that fails to meet these. provisions.

Your hospital was surveyed on June 27, 2003, by the Alabama State Survey Agency based on an allegation of noncompliance with the requirements of 42 CFR 489.24, Responsibilities of Medicare Participating Hospitals in Emergency Cases. After a careful review of the findings, we have determined that your hospital violated the requirements of 42 CFR 489.24 based on:

- Failure to avoid delay in providing a medical screening exam in order to inquire about the individual's method of payment or insurance status for an individual who came to the hospital requesting services.

The deficiencies identified are listed on the enclosed form CMS-2567 Statement of Deficiencies.

We have determined that the deficiencies are so serious that they constitute an immediate threat to the health and safety of any individual who comes to the emergency department and requests examination or treatment for an emergency medical condition. Further, under 42 CFR 480.53, a hospital that violates the provisions of 42 CFR 489.24 is subject to termination of its provider agreement. Consequently, we plan to terminate Huntsville Hospital's participation in the Medicare program

This preliminary determination letter serves to notify you of the violation. The projected date on which your agreement will terminate is September 2, 2004.

You will receive a notice of "termination letter" on August 29, 2004. This final notice will be sent to you concurrently with notice to the public, in accordance with regulations at 42 CFR 489.53.

You may avoid termination action and notice to the public either by providing credible allegation or credible evidence of correction of the deficiencies, or by successfully proving that the deficiencies did not exist, prior to the projected public notification date. In either case, the information must he furnished to this office so that there is time to verify the corrections. A credible allegation of correction provided by the hospital requires a resurvey to verify the corrections. However, when evidence of correction is provided by the hospital, this office must decide whether the evidence of correction is sufficient to halt the termination action. If the evidence is not sufficient in itself to establish that the hospital is in compliance, a resurvey is required for verification of correction. If we verify your corrective action or determine that you successfully refuted the findings contained in this letter by proving that allegations were in error, your

termination from the Medicare program will be rescinded.

Your written response with the corrective actions must be received in our office no later than ten (10) calendar days from receipt of this notice.

In addition to the above actions, CMS is closing the following cases from complaint investigations where we found no violations: complaint control # 070604 and 070904 both with survey date of July 14, 2004.

If you have any questions concerning this preliminary determination letter, please contact Ann Pfeiffer Huffman at 404-562-7462 or Colleen Sandmann at 404-562-7458.

Sincerely,

Sandra M. Pace
Associate Regional Administrator
Division of Survey and Certification

Enclosures:
CMS-2567 Statement of Deficiencies

cc: State Agency Title XVIII
OIG
OCRTO
Complainant

SMP/aph

APPENDIX F

May 15, 2003

Professor W. Sherman Rogers
Howard University School of Law
405 Houston Hall
2900 Van Ness Street, N.W.
Washington, DC 20008

RE: Jeannette Rogers Dulan v. Huntsville Hospital

Dear Professor Rogers:

The following reflects my initial expert opinion regarding the treatment of Professor Dulan at Huntsville Hospital. This opinion is based on the medical records provided by the hospital, as well as the statements of Professor Dulan and others. I would be happy to update my impression should additional information become available.

At the time of the event under consideration, Professor Dulan was 54 years old. She had a history of hypertension, was post-menopausal, and had a family history of coronary heart disease. At approximately noon on Friday October 6, 2000, Professor Dulan began to experience a severe "gripping tightness" in her central chest. This was accompanied by profound weakness and diaphoresis (sweating). Recognizing that this might be a cardiac problem, she immediately sought medical treatment in the Emergency Department of Huntsville Hospital. According to Professor Dulan and her mother (who accompanied her), she arrived at the hospital at approximately 12:30 pm, 30 minutes after the onset of her symptoms. Professor Dulan was taken to the registration desk, where she was asked to fill out the registration forms. Despite complaining of severe chest pain and being in

obvious distress, she was then sent back to fill out additional insurance paperwork.

Professor Dulan was eventually assessed by the triage nurse beginning at 1:02 pm. The nurse noted in her assessment that the patient was complaining of persistent, severe, mid-sternal chest tightness, rated as 8 on a scale of 1 to 10. During this assessment, Professor Dulan became nauseous and vomited in the Emergency Department. An electrocardiogram (ECG) was performed at 1:38 pm, approximately 1 hour and 8 minutes after Professor Dulan's arrival at the hospital and 36 minutes after her assessment in triage. The computerized reading of the ECG read exactly as follows: "Marked sinus bradyeardia. Minimal voltage criteria for LVH, may be normal variant. ST elevation, consider early repolarization, pericarditis, or injury. ST abnormality, possible digitalis effect. Abnormal ECG." Blood was drawn for laboratory assessment at 2:18 pm.

Dr. Andershock, the ED attending physician, briefly interviewed and examined Professor Dulan at 2:40 pm, 1 hour and 38 minutes after she was seen in triage and 1 hour and 2 minutes after the first ECG was performed. He noted in the medical record that the patient was a 54-year-old woman with a history of hypertension. She was complaining of constant chest pain in the center of the chest with radiation to the left arm and back, associated with shortness of breath and diaphoresis. He (inaccurately) recorded that her pain began at approximately 1:00 pm. He noted that Professor Dulan's pain was not relieved with antacids. Her pain was noted as "mild", but Dr. Andershock did not mark the level of pain on the 1-10 scale that is printed on the form. Dr. Andershock's full, written interpretation of the ECG was "LVH." He noted that the blood work revealed a CK of 67 and a Troponin of 0; but did not record the myoglobin level (which presumably was available at the same time as the other labs). His clinical impression was marked as "Chest pain, acute, precordial" and

"Abnormal EKG." He marked "Admitted" for the disposition and "Stable" for condition.

The blood work revealed a Troponin <0.01 and a CK-MB of 1.5, both of which are in the normal range. The myoglobin, however, was 75.4, which is significantly above the normal range at Huntsville Hospital (25-58).

Professor Dulan was treated with aspirin at 3:10 pm, approximately 2 hours after presenting to triage. She was also given nitroglycerine paste and morphine, without relief of her ongoing pain.

The cardiology service was eventually contacted, as indicated in the nursing flowsheet at 5:15 pm: "Cardiology to see patient ASAP." Libby Levson, a nurse practitioner who works with Dr. Carl Gessler Jr. (a cardiologist), came to the ED at approximately 5:30 pm. Therefore, this was at a minimum 41/2 hours after Professor Dulan was seen in triage. In Ms. Levson's note, (without documentation of the time and signed by her supervising cardiologist Dr. Carl Gessler, Jr.), Ms. Levson's interpretation of the ECG was "sinus bradycardia with ST elevations in V1 through V5." The impression was "chest pain, questionable unstable angina." The written plan consisted of "admit to CCU on routine orders. Heparin. Nitrates. Aspirin. Plavix, and Pepcid. Rule out myocardial infarction. Cardiac echo. Left heart catheterization, possible PTCA on Monday." Ms. Levson's input apparently led to the initiation of treatment with heparin and intravenous nitroglycerine at 5:35 pm.

Dr. Gessler's time of arrival in the ED department is not noted in the records provided, but was approximately 1 hour after Ms. Levson's arrival. Apparently Dr. Gessler immediately apprehended the severity of the situation and began arrangements for an emergency cardiac catheterization. Professor Dulan was taken to the Coronary Care Unit at 7:20 pm and then to the catheterization laboratory at 8:39 pm. In

his printed report of this procedure, Dr. Chen (the interventional cardiologist) noted that "unfortunately by this time she was already nine hours after onset of symptoms." Dr. Chen then performed a diagnostic angiogram that revealed a total occlusion of the mid left anterior descending (LAD) artery. A left ventriculogram revealed "evidence of apical and inferoapical and distal anteroapical akinesis" with an ejection fraction of 40%. A stenting procedure was performed on the LAD, with 0% residual stenosis but TIMI H (suboptimal) flow.

Professor Dulan remained in Huntsville Hospital until October 11, 2000, at which time she was discharged home.

The treatment Professor Dulan received at Huntsville Hospital clearly falls far short of accepted standards of care. Professor Dulan presented with a classic acute anterior ST-segment elevation myocardial infarction, which is a true medical emergency. There is ample evidence in the medical record, however, that her condition was not treated as such.

I would first like to describe the accepted standard of care, as outlined in the American Heart Association (AHA)/American College of Cardiology (ACC) Guidelines, for a patient such as Professor Dulan, in order to contrast this with the care she actually received. Immediately upon arrival in the ED and stating that she is having chest pain, the patient should be taken to a treatment room and an ECG should be performed. No time should be wasted filling out forms. This ECG must be performed and interpreted by a qualified physician within 10 minutes of the patient's arrival. If there is any suspicion of cardiac ischemia, the patient should immediately be given aspirin. If the ECG demonstrates ST elevation – a medical emergency - the patient should immediately be considered for reperfusion therapy to open the blocked artery. If a qualified practitioner is available, emergency angioplasty should be performed, with a "door-to-balloon time" of no more than 90 minutes. Another

reasonable alternative treatment is to use a fibrinolytic agent ("clot-buster"), which should be given within 30 minutes of the patient's arrival in the ED.

In Professor Dulan's case, none of these standards of care was followed. First, clear, universally-accepted guidelines are provided by the American Heart Association and the American College of Cardiology for the initial evaluation and treatment of all patients presenting to the emergency department with chest pain. All patients with acute chest pain suspicious for cardiac ischemia should be immediately evaluated. There is absolutely no doubt that Professor Dulan had a history and symptoms consistent with cardiac ischemia. She was a post-menopausal, hypertensive woman with the abrupt onset at rest of substernal chest pain radiating to the left arm. This pain was severe and unremitting, was associated with sweating, weakness, nausea, and vomiting. This is literally a textbook example of the presentation of a heart attack. However, despite these classic symptoms, precious time was wasted having her register and fill out insurance paperwork. Specifically, according to the AHA/ACC Guidelines for patients with chest pain, an electrocardiogram should be performed and reviewed by a physician within 10 minutes of the patient's arrival in the ED. In Professor Dulan's case, the first ECG was

treatment depends not only on the amount of time saved but when it occurs. Available data suggest that time saved within the first 1 to 2 hours has greater biological importance than time saved during the later stages of acute MI (5,7,27,28,30). Several randomized trials of prehospital-initiated thrombolysis have advanced our understanding of the impact of early treatment (5,31-34). Acquisition of ECGs in the field and use of a chest-pain checklist (Table 1) leads to more rapid prehospital and hospital care (5,26). Although none of the individual trials showed a reduction in mortality with prehospital-initiated thrombolytic therapy, a meta-analysis of all available trials demonstrated a 17% relative improvement in outcome associated with prehospital therapy (95% confidence interval [CI], 2% to 29%) (34). The greatest improvement in outcome is observed when treatment can be initiated in the field 60 to 90 minutes earlier than in the hospital (5,33-35).

Although prehospital-initiated thrombolytic therapy results in earlier treatment, the time savings can be offset in most cases by an improved hospital triage with resultant "door-to-needle time" reduced to 30 minutes or less (4). However, only a small percentage (5% to 10%) of patients with chest pain in the prehospital setting have acute MI and are eligible for thrombolytic therapy (5,25,36). Ensuring proper selection of patients for therapy can be difficult, and avoiding therapy when it is contraindicated has important medical, legal, and economic implications. For these reasons, a general national policy of prehospital thrombolytic therapy cannot currently be advocated. However, in special settings in which physicians are present in the ambulance or prehospital transport times are 90 minutes or longer, this therapeutic strategy should be considered. Observations from prehospital trials suggest that prehospital systems should focus on early diagnosis (a relatively minor augmentation in prehospital services) instead of delivery of therapy.

III. INITIAL RECOGNITION AND MANAGEMENT IN THE EMERGENCY DEPARTMENT

Recommendation

Class I

1. **Emergency department acute MI protocol that yields a targeted clinical examination and a 12-lead ECG within 10 min and a door-to-needle time that is <30 min.**

Detection/Quantification and Risk Assessment

Physicians evaluating patients in the ED for possible admission to the coronary care unit (CCU) face the difficult task of avoiding unnecessary admissions but also minimizing the number of patients discharged home inappropriately. Certain subgroups of patients are known to present with unusual symptoms of acute MI. Women often experience atypical ischemic-type chest discomfort (37), while the elderly may complain of shortness of breath more frequently than ischemic-type chest discomfort (25). In addition, with the advent of reperfusion therapy and the desire to minimize door-to-needle time for administration of thrombolytic agents or rapid triage to the catheterization laboratory for primary PTCA, there is a clear need for better methods of prompt identification of patients experiencing a true acute MI as accurately and as soon as possible. The ECG and a history of ischemic-type chest discomfort remain the primary methods for screening patients for myocardial ischemia and infarction. The 12-lead ECG in the ED is at the center of the decision pathway because of the strong evidence that ST-segment elevation identifies patients who benefit from reperfusion therapy. In patients with ischemic-type chest discomfort, ST-segment elevation on the ECG has a specificity of 91% and a sensitivity of 46% for diagnosing acute MI (38). Mortality increases with the number of ECG leads showing ST elevation (39). Current data do not support administration of thrombolytic agents to patients without ST elevation or BBB, and the benefit of primary PTCA remains uncertain in this population. However, it remains important to admit such patients to the hospital for medical therapy and possible cardiac catheterization (Figure 1).

Initial errors in ECG interpretation can result in up to 12% of patients being categorized inappropriately (ST elevation vs. no elevation), demonstrating a potential benefit of accurate computer-interpreted electrocardiography and facsimile transmission to an expert. Other decision aids such as high-risk clinical indicators (40,41), rapid determination of cardiac serum markers (42,43), two-dimensional echocardiographic screening for regional wall motion abnormalities (44), myocardial perfusion imaging (45), and computer-based diagnostic aids (46,47) are of greatest importance in patients in whom the ECG is nondiagnostic. Two-dimensional echocardiography (transthoracic and transesophageal) is of particular value for rapid triage decisions in patients suspected of having an aortic dissection. Because lethal ventricular arrhythmias may develop abruptly in patients with an acute coronary syndrome, all patients should be monitored electrocardiographically on arrival in the ED. It is important to examine serial tracings during evaluation in the ED for development of ST elevation, an event that may be detected by intermittent visual inspection of the oscilloscope or auditory alarms in systems with continuous ST-monitoring capability.

All patients with complicated infarctions (eg, cardiogenic shock) and/or those requiring sophisticated, labor-intensive treatments (eg, intra-aortic balloon counterpulsation) should be admitted to the CCU. In many hospitals physicians admit low-risk MI patients to a coronary observation unit or telemetry unit where electrocardiographic monitoring and defibrillation equipment are available, but other forms of monitoring are not, and staffing is reduced.

According to the World Health Organization (WHO) definition, the diagnosis of MI is based on the presence of at least two of the following three criteria: (1) a clinical history of ischemic-type chest discomfort, (2) changes on serially obtained electrocardiographic tracings, and (3) a rise and fall in serum cardiac markers (10,48). Approximately 70% to 80% of patients with MI present with ischemic-type chest discomfort (49,50). Conversely, less than 25% of patients admitted to the hospital with ischemic-type chest discomfort are subsequently diagnosed as having had an acute MI (51,52). Although ST-segment elevation and/or Q waves on the ECG are highly indicative of MI, about 50% of patients with MI do not exhibit ST elevation (53) but display other or nondiagnostic ECG changes (54). Thus, for the majority of patients, the laboratory plays an essential role in establishing the diagnosis of MI (Figure 2).

Serum Cardiac Markers

When myocytes become necrotic, they lose membrane integrity,

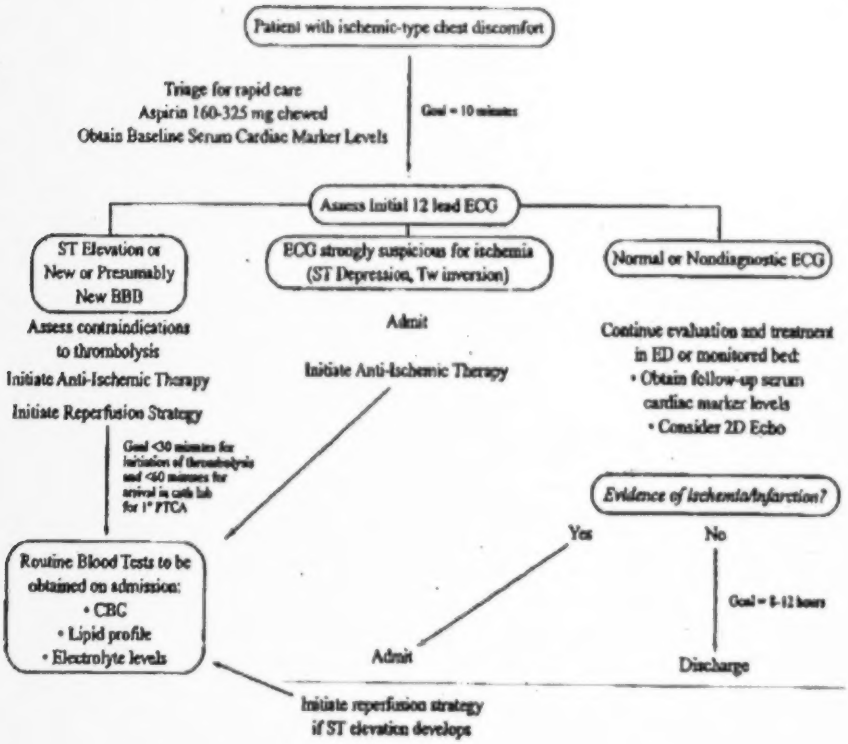

Patient with ischemic-type chest discomfort
Triage for rapid care
Aspirin 160-325 mg chewed
Obtain Baseline Serum Cardiac Marker Levels
Goal = 10 minutes
Assess Initial 12 lead ECG
ST Elevation or New or Presumably New BBB
ECG strongly suspicious for ischemia (ST Depression, Tw inversion)
Normal or Nondiagnostic ECG
Assess contraindications to thrombolysis
Initiate Anti-Ischemic Therapy
Initiate Reperfusion Strategy
Admit
Initiate Anti-Ischemic Therapy
Continue evaluation and treatment in ED or monitored bed:
• Obtain follow-up serum cardiac marker levels
• Consider 2D Echo
Goal <30 minutes for initiation of thrombolysis and <60 minutes for arrival in cath lab for 1° PTCA
Evidence of ischemia/infarction?
Yes
No
Goal = 8-12 hours
Routine Blood Tests to be obtained on admission:
• CBC
• Lipid profile
• Electrolyte levels
Admit
Discharge
Initiate reperfusion strategy if ST elevation develops

Figure 1. Algorithm for management of patients with suspected acute myocardial infarction in the emergency department (ED). All patients with ischemic-type chest discomfort should be evaluated rapidly and receive aspirin. The initial 12-lead electrocardiogram (ECG) is used to define the acute management strategy. Patients with ST-segment elevation or new or presumably new bundle branch block (BBB) should be considered candidates for reperfusion; those without ST-segment elevation but with an ECG and clinical history that are strongly suspicious for ischemia should be admitted for initiation of anti-ischemic therapy (see Fig 4). Patients with a normal or nondiagnostic ECG should undergo further evaluation in the ED or short-term observation until results of serial serum cardiac marker levels are obtained. The following routine blood tests should be obtained in all patients admitted: a complete blood count (CBC), lipid profile, and electrolyte levels. Tw indicates T wave; PTCA, percutaneous transluminal coronary angioplasty. Adapted from Antman EM, Braunwald E. Acute myocardial infarction. In: Braunwald EB, ed. *Heart Disease: A Textbook of Cardiovascular Medicine*. 1996, Philadelphia, PA: WB Saunders.

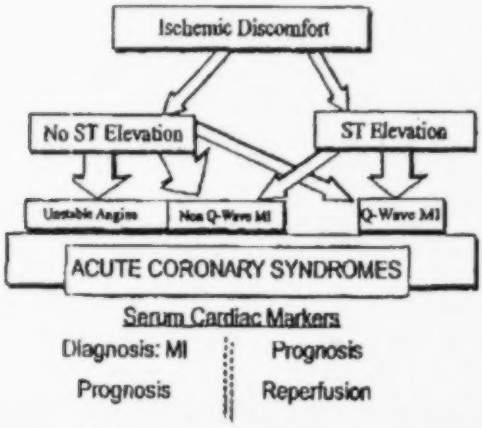
and intracellular macromolecules diffuse into the cardiac interstitium and ultimately into the cardiac microvasculature and lymphatics (55). Eventually, these macromolecules are detectable in the peripheral circulation. The term currently used to collectively describe these macromolecules is serum cardiac markers. An ideal serum cardiac marker of MI should be present early and in high concentration in the myocardium and should be absent from nonmyocardial tissue and serum (55-57). It should be rapidly released into the blood at the time of the myocardial injury, and there should be a stoichiometric relation between the plasma level and the extent of myocardial injury. The marker should persist in blood for a sufficient length of time to provide a convenient diagnostic time window. Finally, measurement of the marker should be easy, inexpensive, and rapid.

The nomenclature of the acute coronary syndromes (ACS) is illustrated in revised Figure 2. The central position of the 12-lead electrocardiogram (ECG) and initial triage of patients are emphasized. Listed at the bottom of the figure is the information sought by clinicians when measuring serum cardiac marker levels in patients at different ends of the ACS spectrum. Serum cardiac markers are useful for confirming the diagnosis of MI when patients present without ST-segment elevation, when the diagnosis

Table 2. Serum Markers of Acute Myocardial Infarction

		Cardiac Troponins			
	Myoglobin	cTnI	cTnT	CK-MB	MB-Isoforms
Molecular Weight (kD)	17	23	33	86	86
First detectable (h)	1-2	3-4		3-4	2-4
100% sensitivity (h)	4-8	8-12		8-12	6-10
Peak (h)	4-8	10-24		10-24	6-12
Duration (d)	(0.5-1.0)	5-10	5-14	2-4	0.5-1.0

cTnI indicates cardiac specific troponin I; cTnT, cardiac specific troponin T. Adapted with permission from Adams J, Abendschein D, Jaffe A. Biochemical markers of myocardial injury: is MB creatine kinase the choice for the 1990s? Circulation. 1993;88:750-763



Revised Figure 2. Patients with ischemic discomfort may present with or without ST-segment elevation on the electrocardiogram. The majority (large arrow) of patients with ST-segment elevation ultimately develop a Q-wave acute myocardial infarction (AMI), whereas a minority (small arrow) develop a non-Q-wave AMI. Of patients who present without ST-segment elevation, the majority (large arrows) are ultimately diagnosed as having either unstable angina or non-Q-wave AMI based on the presence or absence of a cardiac marker such as CK-MB detected in the serum; a minority of such patients ultimately develop a Q-wave AMI. The spectrum of clinical conditions ranging from unstable angina to non-Q-wave AMI and Q-wave AMI is referred to as acute coronary syndromes. AMI indicates acute myocardial infarction. *Adapted from:* Antman EM, Braunwald E. Acute myocardial infarction. In: Braunwald EB, editor. Heart Disease: A Textbook of Cardiovascular Medicine, 1996, Philadelphia, PA: WB Saunders.

may be unclear, and when clinicians must distinguish patients with unstable angina from those with a non–Q-wave MI. Serum cardiac markers also provide valuable prognostic information. For patients with ST-segment elevation, the diagnosis of MI is secure; clinicians are interested in prognostic information as well as a noninvasive assessment of the likelihood that the patient has undergone successful reperfusion when thrombolytic therapy is administered.

Because the conventional serum cardiac marker, creatine kinase (CK) and its MB isoenzyme (CK-MB) lack sufficient sensitivity and specificity, there is a need for more sensitive and cardiac-specific markers of myocardial necrosis (792-794). The troponin complex consists of 3 subunits: troponin T, troponin I, and troponin C (793). The ternary troponin complex is a calcium-sensitive molecular apparatus that regulates the interaction of actin and myosin. Troponin T binds the troponin complex to tropomyosin, and troponin I binds to actin and inhibits interactions between actin and myosin. Troponin C is responsive to changes in intracellular calcium concentration. Amino acid sequences of the skeletal and cardiac isoforms of troponin I and troponin T have sufficient dissimilarity that monoclonal antibody-based immunoassays have been developed to detect cardiac-specific troponin T (cTnT) and cardiac-specific troponin I (cTnI). Because the amino acid sequence of troponin C is the same in cardiac and skeletal muscle, no immunoassays of troponin C have been developed for clinical purposes.

Because CK-MB is found in the skeletal muscle and blood of healthy subjects, the cutoff value for an elevated CK-MB level is typically set a few units above the upper end of the reference (normal) range. In contrast, because cardiac troponin I and cardiac troponin T are not normally detected in the blood of healthy people, the cutoff value for elevated cTnI and cTnT levels may be set only slightly above the noise level of the assay, permitting clinicians to diagnose lesser degrees of myocardial necrosis (ie, increased sensitivity) (796). Because CK and CK-MB are characteristically used as the gold standard for diagnosing MI, investigators may face a dilemma when a new diagnostic test is more sensitive than the gold standard, particularly for identifying episodes of minor myocardial cell necrosis. Case reports confirm histologic evidence of focal myocyte necrosis in patients with elevated cardiac troponin levels and normal CK values (796). It is estimated that ≈30% of patients presenting without ST-segment elevation who would otherwise be diagnosed with unstable angina are actually experiencing a non–Q-wave MI when assessed with cardiac-specific troponin assays (797). Furthermore, numerous investigators have now reported that elevated levels of cTnI or cTnT provide more prognostic information than that supplied by the patient's demographic characteristics or the ECG at presentation (798,799). Elevated cTnI or cTnT levels, even in the presence of normal CK-MB levels, identify patients without ST-segment elevation who are at an increased risk of death. Finally, patients presenting without ST-segment elevation who are characterized as high risk because of elevated cardiac-specific troponin levels demonstrate a greater benefit from treatment with new therapies such as glycoprotein (GP) IIb/IIIa inhibitors than patients without elevated cardiac-specific troponin levels who receive such new pharmacotherapeutic interventions (800).

CK-MB isoforms are another new serum cardiac marker that may be useful for evaluating patients with an acute coronary syndrome. CK-MB exists in only 1 form in myocardial tissue but in different isoforms (or subforms) in the plasma. An absolute level of CK-MB2 >1 U/L or a ratio of CK-MB2 to CK-MB1 of 1.5 has improved sensitivity and specificity for diagnosis of MI within the first 6 hours compared with conventional assays for CK-MB (59). Myoglobin, a low-molecular-weight heme protein found in cardiac and skeletal muscle, is not cardiac specific but is released more rapidly from infarcted myocardium than CK-MB and may be detected as early as 2 hours after MI. The diagnostic sensitivity and specificity for MI were compared for total CK-MB (activity and mass), CK-MB subforms, myoglobin, cTnI, and cTnT in the Diagnostic Marker Cooperative Study (DMCS) (801). The DMCS was a large, prospective, multicenter, double-blind study of patients presenting in the emergency department (ED) with chest pain. CK-MB subforms were most efficient for early diagnosis (within 6 h) of MI, whereas cTnI and cTnT were highly cardiac specific and particularly efficient for late diagnosis of MI. The DMCS investigators concluded that either a single assay (CK-MB subforms) or a select combination (CK-MB subform and a cardiac-specific troponin) reliably triages patients with chest pain and could potentially lead to improved therapy and reduced cost of care of ACS patients. It should be noted that serum levels of cTnT and cTnI may be present for several days after MI (up to 7 days for cTnI and up to 10 to 14 days for cTnT). Therefore, the ability to diagnose recurrent infarction is significantly compromised if the clinician relies solely on cardiac-specific troponins and fails to obtain a concomitant CK or CK-MB measurement within the first 12 to 24 hours of admission of an MI patient. Thus, although CK and CK-MB are not as cardiac specific as the troponins, they will return to normal levels within the first 24 to

36 hours, making it more likely that a reelevation is associated with recurrent myocardial necrosis. For patients presenting within the first two or three hours of symptom onset, the two markers most appropriate for the early diagnosis of AMI are myoglobin and CK-MB subforms.

In patients presenting with ST-segment elevation, clinicians usually use peak CK as a rough estimate of the magnitude of the infarct and assessment of the patient's prognosis. Release of cardiac-specific troponins is stoichiometrically correlated with the amount of myocardial necrosis, and the new serum cardiac markers can also be used to estimate infarct size and prognosis (58). Cardiac-specific troponins may not be detectable for up to 6 hours after onset of chest pain. Thus, when cTnI and cTnT levels are elevated early after onset of discomfort in patients with ST-segment elevation MI, clinicians should suspect that an antecedent episode of unstable angina was in fact MI and the patient is exhibiting a stuttering course of occlusion and release of the infarct-related artery. Data from the Global Utilization of Streptokinase and TPA for Occluded Arteries (GUSTO) III Study suggest that patients with elevated cardiac troponin T levels and who are <6 hours from the onset of discomfort have an increased mortality risk (802).

In addition to monitoring the patient for resolution of ischemic-type chest discomfort and regression of the magnitude of ST-segment elevation on the ECG, clinicians can obtain serial measurements of serum cardiac markers to buttress the noninvasive diagnosis of reperfusion of the infarct-related artery after thrombolytic therapy (65,803). Because of its rapid-release kinetics, myoglobin is a particularly attractive marker for the early diagnosis of reperfusion.

Bedside Testing for Serum Cardiac Markers

Handheld rapid bedside assays are clinically available for measuring cTnI, cTnT, myoglobin, and CK-MB. Small desktop rapid analyzers are also available for the same purpose. A rapid, high-voltage electrophoretic system is available for measuring CK-MB isoforms. When using a handheld rapid bedside assay for a serum cardiac marker, the clinician places a small aliquot of the patient's blood or serum in the specimen well and observes the development of a colored line in the read zone of the device. It should be noted that the time to development of the colored line and the intensity of the color are related to the concentration of the serum cardiac marker in the specimen. For example, when a handheld bedside immunoassay is used to test the blood of patients with high cTnT levels, a red line quickly appears; such patients are at increased mortality risk (804). Careful attention to the timing of the appearance of a positive bedside assay result may provide clinicians with a tool for a semiquantitative estimate of a serum cardiac marker level at the patient's bedside. A positive bedside test however should be confirmed by a conventional quantitative test.

Routine Measures (Oxygen, Nitroglycerin, Aspirin)

Recommendations

Class I

1. **Supplemental oxygen, intravenous access, and continuous electrocardiographic monitoring should be established in all patients with acute ischemic-type chest discomfort.**
2. **An ECG should be obtained and interpreted within 10 min of arrival in the ED in all patients with suspected acute ischemic-type chest discomfort.**

Although the specific diagnosis of acute MI can be made with absolute certainty only occasionally at the time of a patient's entry into the healthcare system, the immediate management of all acute coronary syndromes is generally the same. All patients suspected of having an acute MI should have a clinical and electrocardiographic evaluation that is prompt and targeted to estimate the likelihood that the presenting condition is an acute MI as opposed to one of its potentially lethal mimics: aortic dissection, acute pericarditis, acute myocarditis, spontaneous pneumothorax, or pulmonary embolism.

Although local settings vary widely, the entry process should be completed by a health team member (or members) with the competency to make such an assessment within a very short time of the patient's presentation, ideally within the first 10 minutes and certainly no more than 20 minutes from presentation. Only then should specific procedures or therapies be given, except for securing peripheral venous access. At this entry stage it is important that all members of the healthcare team interact with the patient and family in a warm and caring fashion while projecting professionalism and confidence.

Oxygen

Recommendations

Class I

1. **Overt pulmonary congestion.**
2. **Arterial oxygen desaturation (SaO_2) <90%.**

Class IIa

1. **Routine administration to all patients with uncomplicated MI during the first 2 to 3 h.**

Class IIb

1. **Routine administration of supplemental oxygen to patients with uncomplicated MI beyond 3 to 6 h.**

It has become universal practice to administer oxygen, usually by nasal prongs, to virtually all patients suspected of having acute ischemic-type chest discomfort, although it is not known whether this therapy limits myocardial damage or reduces morbidity or mortality. If oxygen saturation monitoring is used, therapy with supplemental oxygen is indicated if the saturation is less than 90%. Experimental results indicate that breathing oxygen may limit ischemic myocardial injury (66), and there is evidence oxygen administration reduces ST-segment elevation in patients with MI as well (67). The rationale for use of oxygen is based on the observation that even with uncomplicated MI, some patients are modestly hypoxemic initially, presumably because of ventilation-perfusion mismatch and excessive lung water (68).

In patients with severe CHF, pulmonary edema, or a mechanical complication of acute MI, significant hypoxemia may not be corrected with supplemental oxygen alone. Continuous positive-pressure breathing or endotracheal intubation and mechanical ventilation are often required in such cases and should not be unnecessarily delayed (69). A variety of mechanical ventilators are available, and multiple modes are possible. For patients who do

not have a depressed sensorium and are capable of initiating spontaneous ventilation, the preferred modes to use include intermittent mandatory ventilation, assist control, or pressure-support ventilation (70).

For patients without complications, it should be recalled that excess administration of oxygen can lead to systemic vasoconstriction, and high flow rates can be harmful to patients with chronic obstructive airway disease. On the other hand, because administration of nitroglycerin dilates the pulmonary vascular bed and increases ventilation-perfusion abnormalities, it is reasonable to provide supplemental oxygen, at least in the initial hours, for all patients suspected of having an acute MI. In the absence of compelling evidence for established benefit in uncomplicated cases and in view of its expense, there appears to be little justification for continuing its routine use beyond 2 to 3 hours.

Nitroglycerin

Recommendations for Intravenous Nitroglycerin

Class I

1. **For the first 24 to 48 h in patients with acute MI and CHF, large anterior infarction, persistent ischemia, or hypertension.**
2. **Continued use (beyond 48 h) in patients with recurrent angina or persistent pulmonary congestion.**

Class IIa

None.

Class IIb

1. **For the first 24 to 48 h in all patients with acute MI who do not have hypotension, bradycardia, or tachycardia.**
2. **Continued use (beyond 48 h)* in patients with a large or complicated infarction.**

Class III

1. **Patients with systolic pressure <90 mm Hg or severe bradycardia (<50 bpm).**

Considering that the use of nitrates in acute MI was believed to be contraindicated until the early 1970s (71), it is rather striking that today, with the exception of hypotensive patients, virtually all patients with acute ischemic syndromes will receive at least 1 sublingual nitroglycerin tablet before admission to the hospital. Aside from its known clinical benefit in alleviating ischemic myocardial pain, nitroglycerin is now appreciated as having a dilatory effect on the vascular smooth muscle in vessels throughout the body. Thus, vasodilation of the coronary arteries themselves (especially at or adjacent to sites of recent plaque disruption), the peripheral arteries, and the venous capacitance vessels is particularly beneficial to the patient with acute infarction. However, inadvertent systemic hypotension with resulting worsening of myocardial ischemia is the most serious potential complication of nitroglycerin therapy. Thus, patients with ischemic-type chest discomfort should receive sublingual nitroglycerin unless the initial systolic blood pressure is less than 90 mm Hg. It should be avoided in the presence of marked bradycardia (< 50 bpm) or tachycardia (72) and used with extreme caution, if at all, in patients with suspected right ventricular infarction. Patients with right ventricular infarction are especially dependent on adequate right ventricular preload to maintain cardiac output and can experience profound hypotension during administration of nitrates (73).

Long-acting oral nitrate preparations should be avoided in the early management of acute MI. Sublingual or transdermal nitroglycerin can be used, but intravenous infusion of nitroglycerin allows for more precise minute-to-minute control of this agent. Intravenous nitroglycerin can be successfully titrated by frequent measurement of cuff blood pressure and heart rate. Although invasive hemodynamic monitoring is not mandatory, it may be preferable if high doses of vasodilating agents are required, blood pressure instability ensues, or there is clinical doubt about the adequacy of LV filling pressure. Although quite effective in relieving ischemic-type chest discomfort due to acute coronary syndromes, nitroglycerin should not be used as a substitute for narcotic analgesia that is usually required to manage pain associated with acute MI. For a detailed discussion of the pharmacotherapy and relevant clinical studies pertaining to the use of nitroglycerin in acute MI, see Section V "Rationale and Approach to Pharmacotherapy."

Analgesia

The clinical observation of rapid and complete relief of pain after early reperfusion with thrombolytic therapy reinforces the concept that the pain of acute MI is due to continuing ischemia of viable but jeopardized myocardium rather than the effects of completed myocardial necrosis. Efforts to control pain therefore may reasonably involve use of anti-ischemic interventions, including, in addition to reperfusion, oxygen, nitrates, β-adrenoceptor blocking agents, and, in some circumstances, intra-aortic balloon counterpulsation. Effective analgesia (eg, intravenous morphine) should be administered promptly at the time of diagnosis and should not be delayed on the premise that to do so will obscure ability to evaluate the results of anti-ischemic therapy. See Section IV "Hospital Management" for more detailed discussion of proper analgesia.

Aspirin

Recommendations

Class I

1. **A dose of 160 to 325 mg should be given on day 1 of acute MI and continued indefinitely on a daily basis thereafter.**

Class IIb

1. **Other antiplatelet agents such as dipyridamole, ticlopidine or clopidogrel may be substituted if true aspirin allergy is present or if the patient is unresponsive to aspirin.**

The Second International Study of Infarct Survival (ISIS-2) has shown conclusively the efficacy of aspirin alone for treatment of evolving acute MI with a 35-day mortality reduction of 23% (29). When combined with streptokinase, the reduction in mortality was 42%. A meta-analysis demonstrated that aspirin reduced coronary reocclusion and recurrent ischemic events after thrombolytic therapy with either streptokinase or alteplase (74). In a dose of 160 mg or more, aspirin produces a rapid clinical antithrombotic effect caused by immediate and near-total inhibition of thromboxane A_2 production. Accordingly, aspirin now forms part of the early management of all patients with sus-

*Oral or topical preparations may be substituted

pected acute MI and should be given promptly and certainly within the first 24 hours at a dose between 160 and 325 mg and continued daily indefinitely.

Unlike fibrinolytic agents, there is little evidence for a time-dependent effect of aspirin on early mortality. However, data do support the contention that a chewable aspirin is absorbed more quickly than one swallowed in the early hours after infarction, particularly after opiate therapy. The use of aspirin is contraindicated in those with a hypersensitivity to salicylate and should be used with caution in patients with active ulcer disease. Aspirin suppositories (325 mg) can be used safely and are the recommended route of administration for patients with severe nausea and vomiting or known upper-gastrointestinal disorders. There is currently no evidence that other antiplatelet agents such as dipyridamole, ticlopidine, or sulfinpyrazone have any advantage over aspirin for mortality reduction after acute MI. See Section V "Rationale and Approach to Pharmacotherapy" for additional discussion on the use of aspirin in the management of acute MI, and Section VI "Preparation for Discharge From the Hospital."

Atropine

Recommendations

The following recommendations are applicable from early after onset of acute MI to 6 or 8 hours afterward:

Class I

1. Sinus bradycardia with evidence of low cardiac output and peripheral hypoperfusion or frequent premature ventricular complexes at onset of symptoms of acute MI.
2. Acute inferior infarction with type I second- or third-degree atrioventricular (AV) block associated with symptoms of hypotension, ischemic discomfort, or ventricular arrhythmias.
3. Sustained bradycardia and hypotension after administration of nitroglycerin.
4. For nausea and vomiting associated with administration of morphine.
5. Ventricular asystole.

Class IIa

1. Symptomatic patients with inferior infarction and type I second- or third-degree heart block at the level of the AV node (ie, with narrow QRS complex or with known existing BBB).

Class IIb

1. Administration concomitant with (before or after) administration of morphine in the presence of sinus bradycardia.
2. Asymptomatic patients with inferior infarction and type I second-degree heart block or third-degree heart block at the level of the AV node.
3. Second- or third-degree AV block of uncertain mechanism when pacing is not available.

Class III

1. Sinus bradycardia >40 bpm without signs or symptoms of hypoperfusion or frequent premature ventricular contractions.
2. Type II AV block and third-degree AV block and third-degree AV block with new wide QRS complex presumed due to acute MI.

By its parasympatholytic (anticholinergic) activity, atropine sulfate reduces vagal tone, enhances the rate of discharge of the sinus node, and facilitates AV conduction (75). It may be given as an adjunct to morphine administration when nausea and vomiting occur. During the early moments to hours of acute ischemia or acute MI, atropine is particularly useful in treating sinus bradycardia associated with reduced cardiac output and signs of peripheral hypoperfusion, including arterial hypotension, confusion, faintness, or frequent premature ventricular complexes (76). In this setting, leg elevation and intravenous administration of atropine may be lifesaving.

Atropine for Atrioventricular Block, Sinus Bradycardia, or Ventricular Asystole

Atropine is the drug of choice for the occasional treatment of type I second-degree AV block, especially when complicating inferior MI. It is occasionally useful in third-degree AV block at the AV node level in either restoring AV conduction or enhancing the junctional response. When AV block or sinus bradycardia is associated with CHF, hypotension, or frequent and complex ventricular arrhythmias, atropine may improve AV conduction, increase the sinus rate, and avoid the need for immediate insertion of a transvenous pacemaker (77). As a rule, however, in the absence of hemodynamic compromise, treatment of sinus bradycardia or first- or second-degree AV block is not indicated. Similarly, atropine is rarely, if ever, the drug of choice for management of type II second-degree AV block. On occasion, while failing to improve AV block, atropine may increase the sinus rate, and, in fact, enhance the block.

The recommended dosage of atropine for bradycardia is 0.5 to 1.0 mg intravenously (IV), repeated if needed every 3 to 5 minutes to a total dose of no more than 2.5 mg (0.03 to 0.04 mg/kg), the amount that produces complete vagal blockade. Atropine may also be therapeutic in ventricular asystole, for which the recommended dose is 1 mg IV, to be repeated every 3 to 5 minutes (while CPR continues) if asystole persists. The total cumulative dose should not exceed 2.5 mg over 2.5 hours. The peak action of atropine given intravenously is observed within 3 minutes (1).

Side Effects

When administered in doses of less than 0.5 mg or other than intravenously, atropine may produce a paradoxic effect (namely, bradycardia and depression of AV conduction) (78), which is due either to central reflex stimulation of the vagus or a peripheral parasympathomimetic effect on the heart. Urinary retention is not uncommon following administration of atropine and can be deleterious to the patient with acute MI. Repeated administration of atropine may produce adverse central nervous system effects, including hallucinations and fever. Careful dosing and observation after administration of atropine is necessary because the sinus tachycardia that follows may increase ischemia. Rarely, ventricular tachycardia and fibrillation occur after intravenous administration of atropine (79).

Pacing is the treatment of choice for symptomatic bradycardia

not responding promptly to atropine administration.

Risk Stratification and Management of ST-Segment Elevation/Bundle Branch Block Cohort

Newer Concepts

The spectrum of myocardial ischemia consists of patients with clinical presentations that cover the following range of diagnoses: stable angina, unstable angina, MI without ST elevation, and MI with ST elevation. Clinical discrimination among unstable angina, Q wave, and non–Q-wave MI can only be made retrospectively after serial ECGs and serum cardiac markers have been obtained (Figure 2). Patients with ST-segment elevation have a high likelihood of a coronary thrombus occluding the infarct-related artery (80,81). However, not every ST-elevation MI evolves into a Q-wave MI. Angiographic evidence of occlusive coronary thrombus formation may be seen in more than 90% of patients with ST-elevation MI but in only 1% of patients with stable angina and about 35% to 75% of patients with unstable angina or non–Q-wave MI (80-83). Commonly indicated treatment regimens for all acute coronary ischemic syndromes include aspirin, heparin, β-adrenoceptor blockers, and nitrates. Thrombolytic therapy is highly effective in patients with ST elevation or presumably new LBBB (which obscures the electrocardiographic diagnosis of MI)(27) (Figure 3). At the same time, evidence now suggests that thrombolytic therapy is ineffective (for normal or nonspecific electrocardiographic presentations) and possibly even harmful (for ST-depression presentation) in unstable angina and non–ST-elevation MI subgroups (27,84). Figure 4 presents a suggested schema for management of acute MI without ST-segment elevation.

Noninvasive Imaging in the Emergency Department

Screening patients who present with ischemic-type chest discomfort in the ED is an area of clinical and economic importance. Because only 25% or less of patients admitted to the hospital to "rule out" MI actually suffer an MI, accurate screening techniques to identify patients with ongoing necrosis is an important goal. The usefulness of echocardiography in the ED as a means of screening for MI has been validated, but small areas of infarction can be missed, and the age of a regional wall motion abnormality cannot be determined (85-87a). Thallium and sestamibi imaging in the ED are both very good radioisotope screening techniques (85,88,89) and appear to be quite sensitive. However, their use in the ED is still viewed as experimental and is not recommended. In time, the value of noninvasive imaging may further diminish as rapid assays of specific, earlier, and more sensitive serum markers of myocardial necrosis are developed (56,58,60,61).

Thrombolysis

Recommendations

The constellation of clinical features that must be present (although not necessarily at the same time) to serve as standard indications for administration of thrombolytic therapy to patients with acute MI are as follows: (selection of specific thrombolytic agents or regimens is discussed in "Rationale and Approach to Pharmacotherapy.")

Class I

1. **ST elevation (>0.1 mV, two or more contiguous leads),* time to therapy 12 h or less,† age <75 years.**
2. **Bundle branch block (obscuring ST-segment analysis) and history suggesting acute MI.**

Comment: Treatment benefit is present regardless of gender, presence of diabetes, blood pressure (if < 180 mm Hg systolic), heart rate, or history of previous MI (27). Benefit is greater in the setting of anterior MI, diabetes, low blood pressure (< 100 mm Hg systolic), or high heart rate (> 100 bpm). The earlier therapy begins, the better the outcome, with the greatest benefit decidedly occurring when therapy is given within the first 3 h; proven benefit occurs, however, up to at least within 12 h of the onset of symptoms. Benefit is less with inferior acute MI, except for the subgroup with associated right ventricular infarction (ST elevation RV-4) or anterior-segment depression indicative of a posterior current of injury as often occurs with occlusion of a large circumflex coronary artery.

Class IIa

1. **ST elevation,* age 75 years or older.**

Comment: In persons older than 75 years, the overall risk of mortality from infarction is high without and with therapy. Although the proportionate reduction in mortality is less than in patients younger than 75, the absolute reduction results in 10 lives saved per 1000 patients treated in those over 75. The relative benefit of therapy is reduced (27).

Class IIb

1. **ST elevation,* time to therapy > 12 to 24 h.†**
2. **Blood pressure on presentation >180 mm Hg systolic and/or >110 mm Hg diastolic associated with high-risk MI.**

Comment: Generally there is only a small trend for benefit of therapy after a delay of >12 to 24 h, but thrombolysis may be considered for selected patients with ongoing ischemic pain and extensive ST elevation. Risk of intracranial hemorrhage (ICH) is greater when presenting blood pressure is greater than 180/110 mm Hg, and in this situation the potential benefit of therapy must be weighed carefully against the risk of hemorrhagic stroke. Risk of cardiac rupture appeared to increase with prolonged time to therapy in an earlier meta-analysis (90) but was not associated with increased risk of rupture in a later, larger study (91). Generally patients presenting >12 h after symptom onset were excluded from some but not all trials. An attempt to lower blood pressure first (with nitrates, β-adrenoceptor blockers, etc) is recommended but is not of proven benefit in lowering the risk of ICH. Primary PTCA or CABG may be considered if available.

Class III

1. **ST elevation,* time to therapy >24 h,† ischemic pain resolved.**

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* Repeat ECGs recommended during medical observation in suggestive clinical settings when initial ECG is nondiagnostic of ST elevation.

† Time of symptom onset is defined as the beginning of continuous, persistent discomfort that brought the patient to the hospital.

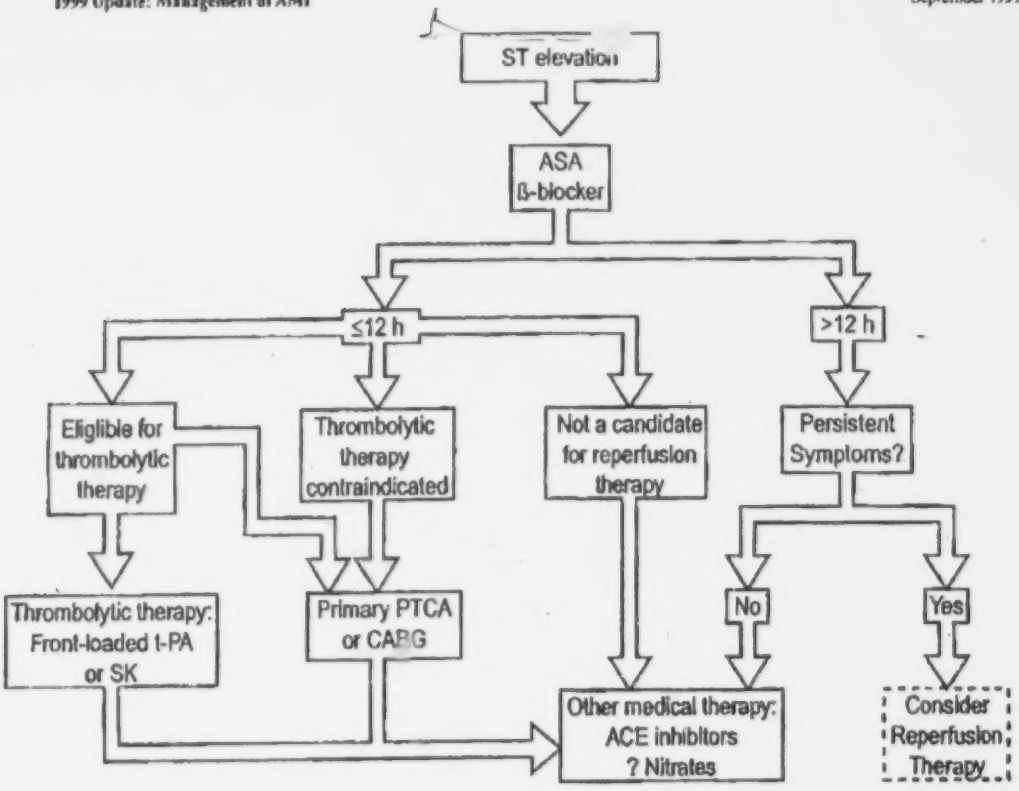


Figure 3. Recommendations for management of patients with ST elevation. All patients with ST-segment elevation on the electrocardiogram should receive aspirin (ASA), ß-adrenoceptor blockers (in the absence of contraindications), and an antithrombin (particularly if tissue-type plasminogen activator [tPA] is used for thrombolytic therapy). Whether heparin is required in patients receiving streptokinase (SK) remains a matter of controversy; the small additional risk for intracranial hemorrhage may not be offset by the survival benefit afforded by adding heparin to SK therapy. Patients treated within 12 hours who are eligible for thrombolytics should expeditiously receive either frontloaded tPA or SK or be considered for primary percutaneous transluminal coronary angioplasty (PTCA). Primary PTCA is also to be considered when thrombolytic therapy is absolutely contraindicated. Coronary artery bypass graft (CABG) may be considered if the patient is less than 6 hours from onset of symptoms. Individuals treated after 12 hours should receive the initial medical therapy noted above and, on an individual basis, may be candidates for reperfusion therapy or angiotensin-converting enzyme (ACE) inhibitors (particularly if left ventricular function is impaired). Modified from Antman EM. Medical therapy for acute coronary syndromes: an overview. In Califf RM, ed. Atlas of Heart Diseases, VIII. Philadelphia, Pa: Current Medicine, 1996.

2. ST-segment depression only.

Comment: In the absence of ST elevation, there is no evidence of benefit for patients with normal electrocardiographic or nonspecific changes, and, using current thrombolytic regimens, there is some suggestion of harm (including increased bleeding risk) for patients with ST-segment depression only (27,92). When marked ST-segment depression is confined to leads V_1 through V4, there is a likelihood that this reflects a posterior current of injury and suggests a circumflex artery occlusion for which thrombolytic therapy would be considered appropriate. Very recent retrospective analysis of the Late Assessment of Thrombolytic Efficacy (LATE) Trial (93,94) also casts some uncertainties about withholding thrombolytic therapy from this heterogeneous group of patients.

A collaborative overview from nine trials of thrombolytic therapy (vs. control) for acute MI has shown a highly significant ($P<0.00001$) 18% proportional reduction in 35-day mortality (9.6% fibrinolysis vs. 11.5% control) corresponding to a reduction of 18 deaths per 1000 patients treated when data from all patient groups are pooled (27). In patients with ST elevation, a proportional mortality reduction of 21% occurred. It is now known that this survival benefit can be maintained long term (6 months to at least 4 years) (27,95).

Figure 5 summarizes the number of lives saved per 1000 patients treated based on the presenting ECG pattern (96). In general, thrombolytic agents should be administered only to patients with ST-segment elevation greater than 0.1 mV or presumably new LBBB on the ECG (27,97). However, in the very early phase of acute infarction, giant, hyperacute T waves may be present with no ST-segment elevation. Similarly, direct posterior infarction can result in ST-segment depressions in leads V_1 through V_3, and in both situations it is appropriate to administer thrombolytic therapy. Thus, it should be clear that certain cases require experienced interpretation of the ECG before withholding reperfusion therapy. Unquestionably, patients with LBBB and anterior ST elevation are at greater inherent risk from MI but also achieve greater benefit with thrombolytic therapy. Although one study (39) suggested that the amount of ST elevation might also predict greater inherent risk and therefore greater benefit, it did not take into account

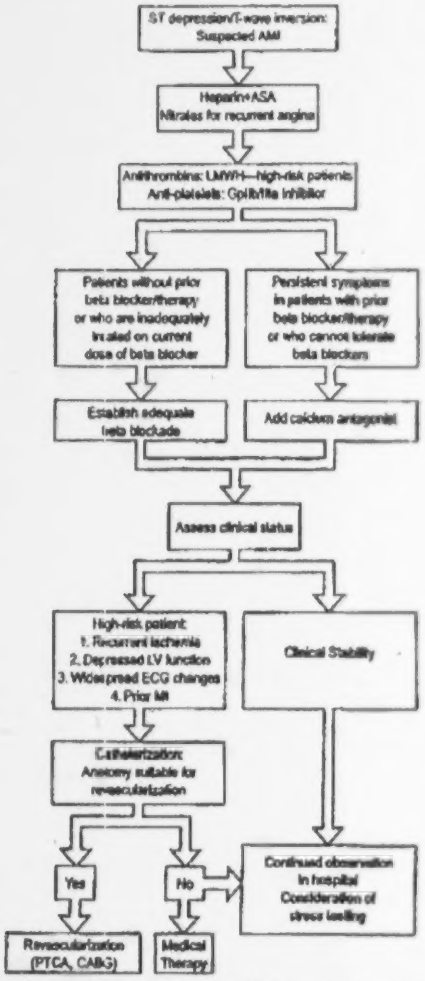

ST depression/T-wave inversion: Suspected AMI
Heparin+ASA
Nitrates for recurrent angina
Antithrombins: LMWH—high-risk patients
Anti-platelets: GpIIb/IIIa Inhibitor
Patients without prior beta blocker/therapy or who are inadequately treated on current dose of beta blocker
Persistent symptoms in patients with prior beta blocker/therapy or who cannot tolerate beta blockers
Establish adequate beta blockade
Add calcium antagonist
Assess clinical status
High-risk patient: 1. Recurrent ischemia 2. Depressed LV function 3. Widespread ECG changes 4. Prior MI
Clinical Stability
Catheterization: Anatomy suitable for revascularization
Yes
No
Continued observation in hospital Consideration of stress testing
Revascularization (PTCA, CABG)
Medical Therapy

Figure 4. All patients without ST elevation should be treated with an antithrombin and aspirin (ASA). Nitrates should be administered for recurrent episodes of angina. Adequate β-adrenoceptor blockade should then be established; when this is not possible or contraindications exist, a calcium antagonist can be considered. Current data indicate that either an invasive or non-invasive treatment strategy is suitable for non-ST-elevation AMI patients. AMI indicates acute myocardial infarction; CABG, coronary artery bypass graft; ECG, electrocardiogram; GpIIb/IIIa, Glycoprotein IIb/IIIa receptor for platelet aggregation; LMWH, low-molecular-weight heparin; LV, left ventricular; PTCA, percutaneous transluminal coronary angioplasty. Modified from Antman EM. Medical therapy for acute coronary syndromes: an overview. In Califf RM, editor. Atlas of Heart Diseases, VIII. Philadelphia, PA: Current Medicine; 1996.

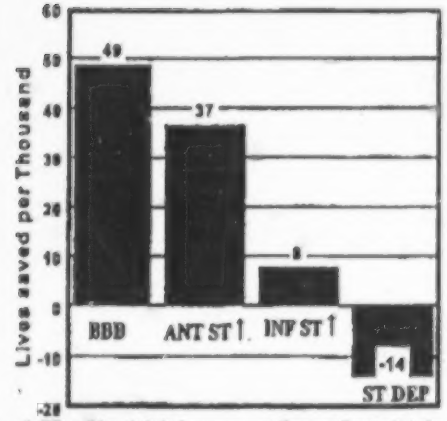


Figure 5. Effect of thrombolytic therapy on mortality according to admission electrocardiogram. Patients with bundle branch block (BBB) and anterior ST-segment elevation (ANT ST↑) derive the most benefit from thrombolytic therapy. Effects in patients with inferior ST-segment elevation (INF ST↑) are much less, while patients with ST-segment depression (ST DEP) do not benefit. Adapted from Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomised trials of more than 1000 patients. Lancet Ltd. 1994;343:311-322. Reprinted from Management of Acute Myocardial Infarction (Julian D, Braunwald E, eds). Martin GV, Kennedy JW. Choice of thrombolytic agent, p 90, 1994, by permission of the publisher, WB Saunders Co Ltd, London.

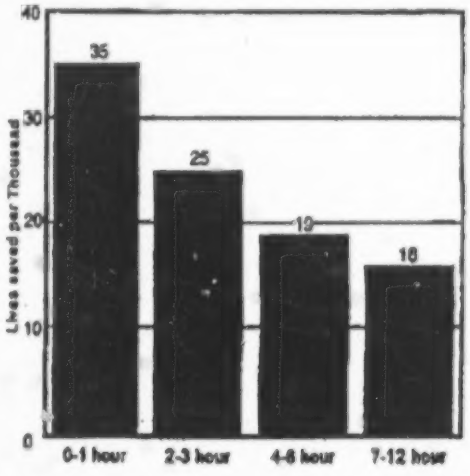


Figure 6. Effect of thrombolytic therapy on mortality according to time from symptom onset. Patients treated early derive the most benefit. Adapted from Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomised trials of more than 1000 patients. Lancet Ltd. 1994;343:311-322. Reprinted from Management of Acute Myocardial Infarction (Julian D, Braunwald E, eds). Martin GV, Kennedy JW. Choice of thrombolytic agent, p 90, 1994, by permission of the publisher, WB Saunders Co Ltd, London.

the increased amount of ST elevation seen in patients with anterior infarction. Other factors such as collateral flow (98) clearly influence the amount of ST elevation, which may limit its value for predicting therapeutic benefit.

Additional factors that influence the decision to administer thrombolytic therapy include time since onset of symptoms, patient's age, hemodynamic status, and coexisting medical illnesses (Figure 6 and Figure 7). Myocardial salvage increases with progressively earlier administration of thrombolytic therapy, although a reduction in mortality may still be seen in patients treated up to at least 12 hours from onset of definitive symptoms (27,99,100). Some patients presenting at more than 12 to 24 hours with persistent ischemic symptoms and ST elevation also may benefit from treatment. Although younger patients achieve a greater relative reduction in mortality compared with older patients, the

New Table 2.1 Intracranial Hemorrhage in Recent Thrombolytic Trials

Patient Characteristics	GUSTO-I (497)	GUSTO-II (805)	GUSTO-III (802, 833)	CORALT (832)	ASSENT-2*	In Time-II*
Number	41,021	3473	15,059	7169	16,950	15,078
Average Age (y)	62	62.5	63	62.4	–	–
>75 y (%)	10.5	11.8	13.6	13.0	–	–
Female (%)	25.2	22.4	27.4	23.4	–	–
Intracranial Hemorrhage Rates						
SK	0.51	0.37	–	–	–	–
tPA	0.70	0.72	0.87	Double bolus 1.12 Accl infusion 0.81	0.93	0.62
rPA	–	–	0.91	–	–	–
TNK-tPA	–	–	–	–	0.94	–
nPA	–	–	–	–	–	1.13

accl indicates accelerated; nPA, lanoteplase; rPA, reteplase; TNK-tPA, a genetically engineered variant of tPA; tPA, tissue plasminogen activator; SK, streptokinase.
*Data based on preliminary results

increasing absolute mortality rates with advancing age result in progressively greater absolute mortality reductions up to age 75. Benefit may also be achieved after age 75 but is less certain than at younger ages (27,101). Advanced age does increase risk of stroke after acute MI, both without and with thrombolytic therapy. Given the much greater mortality risk of MI, the elderly should be considered candidates for thrombolytic therapy after careful screening for exclusions. Patients should be considered at higher risk if they have any of the following: female gender, advanced age (>70 years), history of previous infarction, atrial fibrillation, anterior infarction, rales in more than one third of the lung fields, hypotension, and sinus tachycardia or diabetes mellitus (27,102). Indeed, certain subgroups of patients with an especially high likelihood of benefiting from successful reperfusion include those with hypotension, tachycardia, and a history of diabetes mellitus or prior MI.

Early placebo-controlled trials of thrombolysis for MI raised concern about a paradoxical increase in mortality during the first 24 hours after thrombolysis that was later offset by a greater reduction in mortality in the thrombolytic groups (27). More recently conducted thrombolytic trials have confirmed a "high density" of mortality in the first 24 hours but suggest that this may be attributed primarily to pump failure from unsuccessful reperfusion rather than an early hazard of thrombolysis (103).

Risk of Stroke

Thrombolytic therapy is associated with a slight but definite excess risk of stroke that occurs predominantly within the first day of therapy (27). Clinical variables that can be ascertained in the ED that predict an increased risk of ICH are advanced age (older than 65 years, odds ratio 2.2, 95% CI, 1.4 to 3.5), low body weight (< 70 kg, odds ratio 2.1, CI 1.3 to 3.2), hypertension on presentation (odds ratio 2.0, CI 1.2 to 3.2), and use of alteplase (odds ratio 1.6, CI 1.0 to 2.5) (104-106). The number of risk factors at presentation may be used to estimate the probability of ICH and is shown in Figure 8*. Although no firm guidelines have been established, ICH rates less than 1% have generally been regarded as acceptable in clinical trials, considering the overall favorable benefit-risk profiles, whereas rates greater than 1.5% have been viewed as unacceptably high (107).

* To reduce the risk, the dose of 90-minute alteplase should be adjusted downward for low body weight (<67 kg). Similarly the 180-minute regimen should be adjusted downward for patients who weigh <65 kg.

More recent trials show that as use of thrombolysis has increased, a greater proportion of patients who are >75 years old or female are now included. This change has been associated with a higher rate of ICH than that seen in earlier studies. For example, the rate of ICH after administration of alteplase was ~0.7%; in more recent studies, it is 0.8% to 0.9% (New Table 2.1). It should be noted that the streptokinase without heparin administration regimen has the lowest rate of ICH.

Net Clinical Benefit

Clinicians must carefully weigh the risk-benefit ratio of thrombolysis for individual patients. Hesitancy to prescribe thrombolytic therapy arises from concern about intracranial bleeding and uncertainty about eligibility criteria. The generally higher mortality rate among MI patients who do not undergo thrombolysis underscores the need for heightened awareness of current indications for thrombolysis through such projects as the NHAAP (108). Decision analysis methods suggest that appropriate use of thrombolytic therapy in eligible patients would save many additional lives annually in the United States (109).

Contraindications/Cautions

Hemorrhage represents the most important risk of thrombolytic therapy, especially ICH, which may be fatal in one half to two thirds of patients. Contraindications and cautions to thrombolytic use are given in Table 3.

Summary of Initial Diagnostic and Treatment Strategy

A summary of initial diagnostic and treatment strategies for patients with acute MI with ST elevation or BBB, focusing on emergency management, is provided in Table 4.

Primary Percutaneous Transluminal Coronary Angioplasty

Recommendations

Class I

1. **As an alternative to thrombolytic therapy in patients with AMI and ST-segment elevation or new or presumed new LBBB who can undergo angioplasty of the infarct-related artery within 12 h of onset of symptoms or >12 h if ischemic symptoms persist, if performed in a timely**

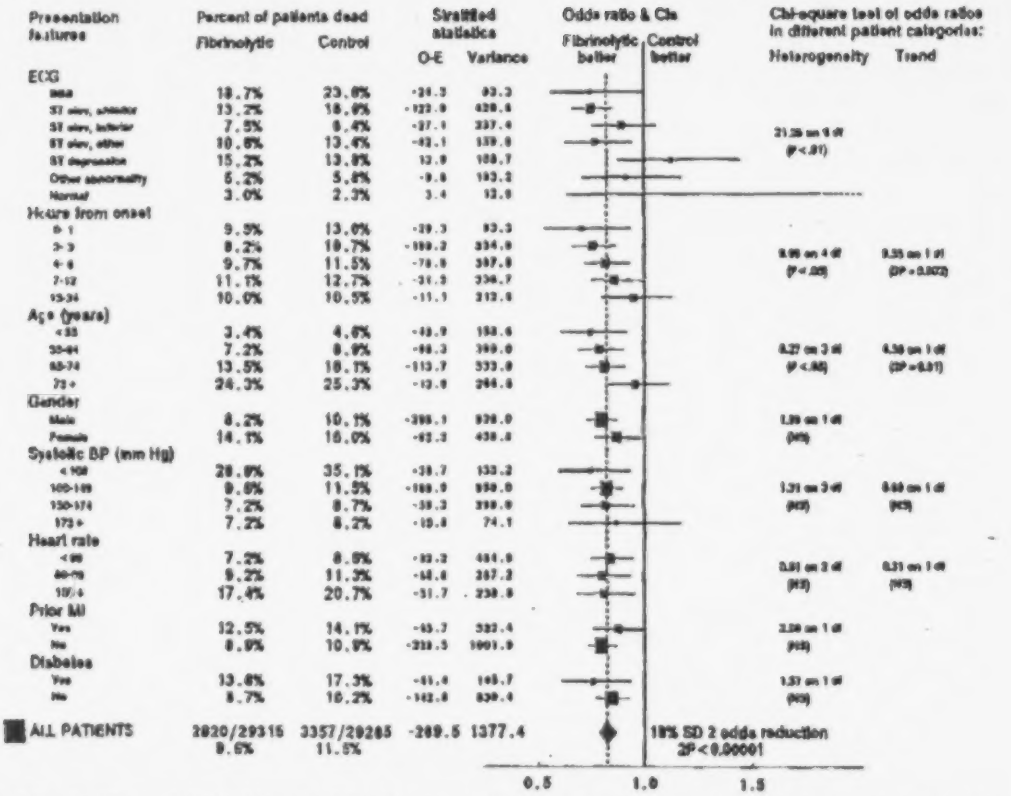


Figure 7. Mortality differences during days 0 through 35 subdivided by presentation features in a collaborative overview of results from nine trials of thrombolytic therapy. At center absolute mortality rates are shown for fibrinolytic and control groups for each clinical feature at presentation listed at left. The odds ratio of death in fibrinolytic group to that in control group is shown for each subdivision (black square) along with 95% confidence interval (horizontal line). The summary odds ratio at bottom corresponds to an 18% proportional reduction in 35-day mortality and is highly statistically significant. This translates to a reduction of 18 deaths per 1000 patients treated with thrombolytic agents. O-E indicates observed versus expected ratio; CIs, confidence intervals; ECG, electrocardiogram; BBB, bundle branch block; ST elev, ST-segment elevation; df, degrees of freedom; BP, blood pressure; MI, myocardial infarction; SD, standard deviation. Adapted from Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomised trials of more than 1000 patients. Lancet Ltd. 1994;343:311-322. Reprinted from Management of Acute Myocardial Infarction (Julian D, Braunwald E, eds). Martin GV, Kennedy JW. Choice of thrombolytic agent, p 42-44, 1994, by permission of the publisher, WB Saunders Co Ltd, London.

fashion* by persons skilled in the procedure† and supported by experienced personnel in an appropriate laboratory environment.‡

2. In patients who are within 36 h of an acute ST-elevation/Q-wave or new LBBB MI who develop cardiogenic shock, are <75 years of age, and revascularization can be performed within 18 h of onset of shock.

Class IIa

1. As a reperfusion strategy in candidates for reperfusion who have a contraindication to thrombolytic therapy (Table 3).

Class IIb

1. In patients with AMI who do not present with ST elevation but who have reduced (less than TIMI [Thrombolysis in Myocardial Infarction] grade 2) flow of the infarct-related artery and when angioplasty can be performed within 12 h of onset of symptoms.

Class III

This classification applies to patients with AMI who:

1. Undergo elective angioplasty of a non–infarct-related artery at the time of AMI
2. Are >12 h after onset of symptoms and have no evidence of myocardial ischemia
3. Have received fibrinolytic therapy and have no symptoms of myocardial ischemia
4. Are eligible for thrombolysis and are undergoing primary angioplasty performed by a low volume operator in a laboratory without surgical capability

Comment: There is serious concern that a routine policy of primary PTCA for patients with acute MI will result in unacceptable delays in achieving reperfusion in a substantial number of cases and less than optimal outcomes if performed by less experienced operators. Strict performance criteria must be mandated for primary angioplasty programs so that

* Performance standard: balloon inflation within 90±30 minutes of admission.
† Individuals who perform >75 PTCA procedures per year (110).
‡ Centers that perform >200 PTCA procedures per year and have cardiac surgical capability (110).

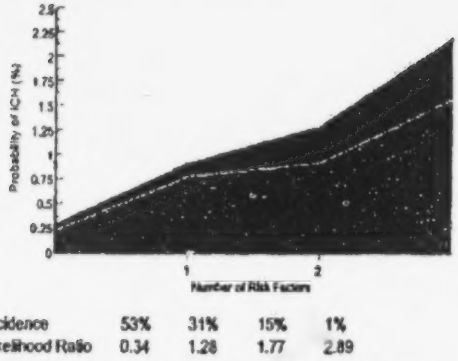


Figure 8. Risk of intracranial hemorrhage (ICH) during thrombolytic therapy. At bottom is estimated incidence of frequency of one or more of the following risk factors: age >65 years, weight <70 kg, hypertension on admission, and use of tissue plasminogen activator (TPA) in patients with acute MI who are potential candidates for thrombolytic therapy. The likelihood ratio describes the probability of finding the risk profile among patients with intracranial bleeding divided by the probability of finding the same risk profile among patients without intracranial bleeding. Curves depict estimated probability of ICH assuming an overall incidence of 0.5% and 0.75% (bottom and top curves respectively). Adapted from data in Simoons ML, Maggioni AP, Knatterud G, et al. Individual risk assessment for intracranial hemorrhage during thrombolytic therapy. Lancet Ltd. 1993;342:1523-1528

Table 3. Contraindications and Cautions for Thrombolytic Use in Myocardial Infarction*

Contraindications
• Previous hemorrhagic stroke at any time; other strokes or cerebrovascular events within 1 year
• Known intracranial neoplasm
• Active internal bleeding (does not include menses)
• Suspected aortic dissection
Cautions/relative contraindications
• Severe uncontrolled hypertension on presentation (blood pressure >180/110 mm Hg)†
• History of prior cerebrovascular accident or known intracerebral pathology not covered in contraindications
• Current use of anticoagulants in therapeutic doses (INR ≥2-3); known bleeding diathesis
• Recent trauma (within 2-4 weeks), including head trauma or traumatic or prolonged (>10 min) CPR or major surgery (<3 wk)
• Non compressible vascular punctures
• Recent (within 2-4 weeks) internal bleeding
• For streptokinase/anistreplase: prior exposure (especially within 5 days-2 y) or prior allergic reaction
• Pregnancy
• Active peptic ulcer
• History of chronic severe hypertension

INR indicates International Normalized Ratio; CPR, cardiopulmonary resuscitation.
*Viewed as advisory for clinical decision making and may not be all-inclusive or definitive.
†Could be an absolute contraindication in low-risk patients with myocardial infarction (see text).

Table 4. Diagnostic and Treatment Measures in Patients With ST Elevation or Bundle Branch Block

Initial diagnostic measures
1. Use continuous ECG, automated BP, HR monitoring
2. Take targeted history (for AMI inclusions, thrombolysis exclusions), check vital signs, perform focused examination
3. Start IV(s), draw blood for serum cardiac markers, hematology, chemistry, lipid profile
4. Obtain 12-lead ECG
5. Obtain chest x-ray (preferably upright)
General treatment measures
1. Aspirin, 160-325 mg (chew and swallow)
2. Nitroglycerin, sublingual: test for Prinzmetal's angina, reversible spasm; anti-ischemic, antihypertensive effects
3. Oxygen: sparse data; probably indicated, first 2-3 h in all; continue if low arterial oxygen saturation (<90%)
4. Adequate analgesia: small doses of morphine (2-4 mg) as needed
Specific treatment measures
1. Reperfusion therapy: goal—door-to-needle time <30 min; door-to-dilation time <60 min
2. Conjunctive antithrombotics: aspirin, heparin (especially with TPA)
3. Adjunctive therapies: β-adrenoceptor blockade if eligible, intravenous nitroglycerin (for anti-ischemic or antihypertensive effects), ACE inhibitor (especially with large or anterior AMI, heart failure without hypotension [SBP >100 mm Hg], previous MI).

ECG indicates electrocardiogram; BP, blood pressure; HR, heart rate; AMI, acute myocardial infarction; IVs, intravenous administration; TPA, tissue plasminogen activator; ACE, angiotensin converting enzyme; SBP, systolic blood pressure.

such delays in revascularization and performance by low-volume operators/centers do not occur. Interventional cardiologists and centers must operate within a specified "corridor of outcomes" to include (1) balloon dilation within 90±30 min of admission and diagnosis of AMI; (2) a documented clinical success rate with Thrombolysis in Myocardial Infarction (TIMI) II through III flow attained in >90% of patients without emergency CABG, stroke, or death; (3) emergency CABG rate <5% among all patients undergoing the procedure; (4) actual performance of angioplasty in a high percentage of patients (85%) brought to the laboratory; and (3) mortality rate <10%. Otherwise, the focus of treatment should be the early use of thrombolytic therapy.

Since publication of the original report of primary (direct) PTCA as an alternative to thrombolytic therapy in patients with acute MI, (111) its merits have been debated (112,113). There are no randomized controlled trials of primary PTCA versus no reperfusion. Thus, the recommendations are based on findings from small and moderately sized comparative trials of primary PTCA and thrombolysis and from indirect evidence.

Initial assessments showed that PTCA restored antegrade flow in the occluded infarct-related artery in more than 90% of patients and was associated with a 1-year survival rate of 90% to 96% (114-117). Subsequently several randomized trials compared PTCA and thrombolytic therapy in patients with acute MI (118-120). In these studies PTCA was reported to successfully restore antegrade coronary flow in approximately 88% to 95% of attempts. In the study of Zijlstra et al, (118) follow-up angiography weeks after infarction showed that the infarct-related artery was patent in 91% of those who had primary PTCA (studied on average 3 months after the procedure) and in 68% of those who received streptokinase (P=.001) (studied 3 weeks later), and the residual infarct-related artery stenosis was less in those who underwent PTCA. Those in whom primary PTCA was performed also had fewer in-hospital adverse events (nonfatal reinfarction or death) and were less likely to have recurrent ischemia or to require coronary revascularization over the period of follow-up.

Similarly, Gibbons et al (119) found that those who underwent

treatment depends not only on the amount of time saved but when it occurs. Available data suggest that time saved within the first 1 to 2 hours has greater biological importance than time saved during the later stages of acute MI (5,7,27,28,30). Several randomized trials of prehospital-initiated thrombolysis have advanced our understanding of the impact of early treatment (5,31-34). Acquisition of ECGs in the field and use of a chest-pain checklist (Table 1) leads to more rapid prehospital and hospital care (5,26). Although none of the individual trials showed a reduction in mortality with prehospital-initiated thrombolytic therapy, a meta-analysis of all available trials demonstrated a 17% relative improvement in outcome associated with prehospital therapy (95% confidence interval [CI], 2% to 29%) (34). The greatest improvement in outcome is observed when treatment can be initiated in the field 60 to 90 minutes earlier than in the hospital (5,33-35).

Although prehospital-initiated thrombolytic therapy results in earlier treatment, the time savings can be offset in most cases by an improved hospital triage with resultant "door-to-needle time" reduced to 30 minutes or less (4). However, only a small percentage (5% to 10%) of patients with chest pain in the prehospital setting have acute MI and are eligible for thrombolytic therapy (5,25,36). Ensuring proper selection of patients for therapy can be difficult, and avoiding therapy when it is contraindicated has important medical, legal, and economic implications. For these reasons, a general national policy of prehospital thrombolytic therapy cannot currently be advocated. However, in special settings in which physicians are present in the ambulance or prehospital transport times are 90 minutes or longer, this therapeutic strategy should be considered. Observations from prehospital trials suggest that prehospital systems should focus on early diagnosis (a relatively minor augmentation in prehospital services) instead of delivery of therapy.

III. INITIAL RECOGNITION AND MANAGEMENT IN THE EMERGENCY DEPARTMENT

Recommendation

Class I

1. **Emergency department acute MI protocol that yields a targeted clinical examination and a 12-lead ECG within 10 min and a door-to-needle time that is <30 min.**

Detection/Quantification and Risk Assessment

Physicians evaluating patients in the ED for possible admission to the coronary care unit (CCU) face the difficult task of avoiding unnecessary admissions but also minimizing the number of patients discharged home inappropriately. Certain subgroups of patients are known to present with unusual symptoms of acute MI. Women often experience atypical ischemic-type chest discomfort (37), while the elderly may complain of shortness of breath more frequently than ischemic-type chest discomfort (25). In addition, with the advent of reperfusion therapy and the desire to minimize door-to-needle time for administration of thrombolytic agents or rapid triage to the catheterization laboratory for primary PTCA, there is a clear need for better methods of prompt identification of patients experiencing a true acute MI as accurately and as soon as possible. The ECG and a history of ischemic-type chest discomfort remain the primary methods for screening patients for myocardial ischemia and infarction. The 12-lead ECG in the ED is at the center of the decision pathway because of the strong evidence that ST-segment elevation identifies patients who benefit from reperfusion therapy. In patients with ischemic-type chest discomfort, ST-segment elevation on the ECG has a specificity of 91% and a sensitivity of 46% for diagnosing acute MI (38). Mortality increases with the number of ECG leads showing ST elevation (39). Current data do not support administration of thrombolytic agents to patients without ST elevation or BBB, and the benefit of primary PTCA remains uncertain in this population. However, it remains important to admit such patients to the hospital for medical therapy and possible cardiac catheterization (Figure 1).

Initial errors in ECG interpretation can result in up to 12% of patients being categorized inappropriately (ST elevation vs. no elevation), demonstrating a potential benefit of accurate computer-interpreted electrocardiography and facsimile transmission to an expert. Other decision aids such as high-risk clinical indicators (40,41), rapid determination of cardiac serum markers (42,43), two-dimensional echocardiographic screening for regional wall motion abnormalities (44), myocardial perfusion imaging (45), and computer-based diagnostic aids (46,47) are of greatest importance in patients in whom the ECG is nondiagnostic. Two-dimensional echocardiography (transthoracic and transesophageal) is of particular value for rapid triage decisions in patients suspected of having an aortic dissection. Because lethal ventricular arrhythmias may develop abruptly in patients with an acute coronary syndrome, all patients should be monitored electrocardiographically on arrival in the ED. It is important to examine serial tracings during evaluation in the ED for development of ST elevation, an event that may be detected by intermittent visual inspection of the oscilloscope or auditory alarms in systems with continuous ST-monitoring capability.

All patients with complicated infarctions (eg, cardiogenic shock) and/or those requiring sophisticated, labor-intensive treatments (eg, intra-aortic balloon counterpulsation) should be admitted to the CCU. In many hospitals physicians admit low-risk MI patients to a coronary observation unit or telemetry unit where electrocardiographic monitoring and defibrillation equipment are available, but other forms of monitoring are not, and staffing is reduced.

According to the World Health Organization (WHO) definition, the diagnosis of MI is based on the presence of at least two of the following three criteria: (1) a clinical history of ischemic-type chest discomfort, (2) changes on serially obtained electrocardiographic tracings, and (3) a rise and fall in serum cardiac markers (10,48). Approximately 70% to 80% of patients with MI present with ischemic-type chest discomfort (49,50). Conversely, less than 25% of patients admitted to the hospital with ischemic-type chest discomfort are subsequently diagnosed as having had an acute MI (51,52). Although ST-segment elevation and/or Q waves on the ECG are highly indicative of MI, about 50% of patients with MI do not exhibit ST elevation (53) but display other or nondiagnostic ECG changes (54). Thus, for the majority of patients, the laboratory plays an essential role in establishing the diagnosis of MI (Figure 2).

Serum Cardiac Markers

When myocytes become necrotic, they lose membrane integrity,

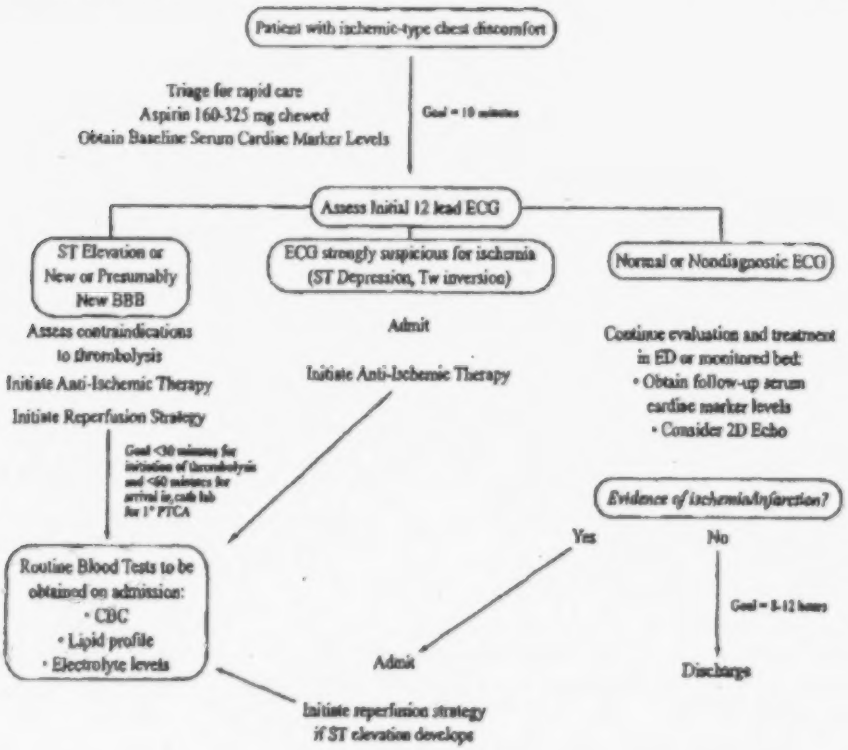

Patient with ischemic-type chest discomfort
Triage for rapid care
Aspirin 160-325 mg chewed
Obtain Baseline Serum Cardiac Marker Levels
Goal = 10 minutes
Assess Initial 12 lead ECG
ST Elevation or New or Presumably New BBB
ECG strongly suspicious for ischemia (ST Depression, Tw inversion)
Normal or Nondiagnostic ECG
Assess contraindications to thrombolysis
Initiate Anti-Ischemic Therapy
Initiate Reperfusion Strategy
Admit
Initiate Anti-Ischemic Therapy
Continue evaluation and treatment in ED or monitored bed:
• Obtain follow-up serum cardiac marker levels
• Consider 2D Echo
Goal <30 minutes for initiation of thrombolysis and <60 minutes for arrival in cath lab for 1° PTCA
Evidence of Ischemia/Infarction?
Yes
No
Routine Blood Tests to be obtained on admission:
• CBC
• Lipid profile
• Electrolyte levels
Goal = 8-12 hours
Admit
Discharge
Initiate reperfusion strategy if ST elevation develops

Figure 1. Algorithm for management of patients with suspected acute myocardial infarction in the emergency department (ED). All patients with ischemic-type chest discomfort should be evaluated rapidly and receive aspirin. The initial 12-lead electrocardiogram (ECG) is used to define the acute management strategy. Patients with ST-segment elevation or new or presumably new bundle branch block (BBB) should be considered candidates for reperfusion; those without ST-segment elevation but with an ECG and clinical history that are strongly suspicious for ischemia should be admitted for initiation of anti-ischemic therapy (see Fig 4). Patients with a normal or nondiagnostic ECG should undergo further evaluation in the ED or short-term observation until results of serial serum cardiac marker levels are obtained. The following routine blood tests should be obtained in all patients admitted: a complete blood count (CBC), lipid profile, and electrolyte levels. Tw indicates T wave; PTCA, percutaneous transluminal coronary angioplasty. Adapted from Antman EM, Braunwald E. Acute myocardial infarction. In: Braunwald EB, ed. *Heart Disease: A Textbook of Cardiovascular Medicine*. 1996, Philadelphia, PA: WB Saunders.

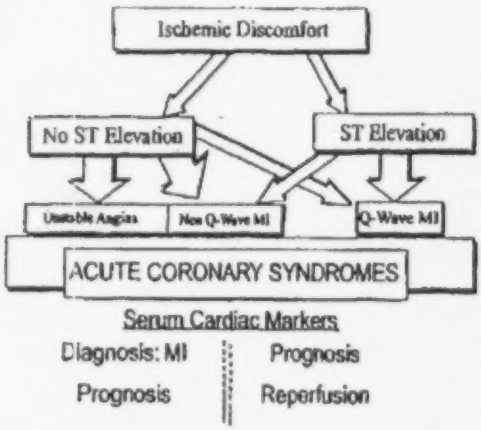
and intracellular macromolecules diffuse into the cardiac interstitium and ultimately into the cardiac microvasculature and lymphatics (55). Eventually, these macromolecules are detectable in the peripheral circulation. The term currently used to collectively describe these macromolecules is serum cardiac markers. An ideal serum cardiac marker of MI should be present early and in high concentration in the myocardium and should be absent from nonmyocardial tissue and serum (55-57). It should be rapidly released into the blood at the time of the myocardial injury, and there should be a stoichiometric relation between the plasma level and the extent of myocardial injury. The marker should persist in blood for a sufficient length of time to provide a convenient diagnostic time window. Finally, measurement of the marker should be easy, inexpensive, and rapid.

The nomenclature of the acute coronary syndromes (ACS) is illustrated in revised Figure 2. The central position of the 12-lead electrocardiogram (ECG) and initial triage of patients are emphasized. Listed at the bottom of the figure is the information sought by clinicians when measuring serum cardiac marker levels in patients at different ends of the ACS spectrum. Serum cardiac markers are useful for confirming the diagnosis of MI when patients present without ST-segment elevation, when the diagnosis

Table 2. Serum Markers of Acute Myocardial Infarction

		Cardiac Troponins			
	Myoglobin	cTnI	cTnT	CK-MB	MB-Isoforms
Molecular Weight (kD)	17	23	33	86	86
First detectable (h)	1-2	2-4		3-4	2-4
100% sensitivity (h)	4-8	8-12		8-12	6-10
Peak (h)	4-8	10-24		10-24	6-12
Duration (d)	(0.5-1.0)	5-10	5-14	2-4	0.5-1.0

cTnI indicates cardiac specific troponin I; cTnT, cardiac specific troponin T. Adapted with permission from Adams J, Abendschein D, Jaffe A. Biochemical markers of myocardial injury: is MB creatine kinase the choice for the 1990s? Circulation. 1993;88:750-763



Revised Figure 2. Patients with ischemic discomfort may present with or without ST-segment elevation on the electrocardiogram. The majority (large arrow) of patients with ST-segment elevation ultimately develop a Q-wave acute myocardial infarction (AMI), whereas a minority (small arrow) develop a non-Q-wave AMI. Of patients who present without ST-segment elevation, the majority (large arrows) are ultimately diagnosed as having either unstable angina or non-Q-wave AMI based on the presence or absence of a cardiac marker such as CK-MB detected in the serum; a minority of such patients ultimately develop a Q-wave AMI. The spectrum of clinical conditions ranging from unstable angina to non-Q-wave AMI and Q-wave AMI is referred to as acute coronary syndromes. AMI indicates acute myocardial infarction. Adapted from: Antman EM, Braunwald E. Acute myocardial infarction. In: Braunwald EB, editor. Heart Disease: A Textbook of Cardiovascular Medicine, 1996, Philadelphia, PA: WB Saunders.

may be unclear, and when clinicians must distinguish patients with unstable angina from those with a non–Q-wave MI. Serum cardiac markers also provide valuable prognostic information. For patients with ST-segment elevation, the diagnosis of MI is secure; clinicians are interested in prognostic information as well as a noninvasive assessment of the likelihood that the patient has undergone successful reperfusion when thrombolytic therapy is administered.

Because the conventional serum cardiac marker, creatine kinase (CK) and its MB isoenzyme (CK-MB) lack sufficient sensitivity and specificity, there is a need for more sensitive and cardiac-specific markers of myocardial necrosis (792-794). The troponin complex consists of 3 subunits: troponin T, troponin I, and troponin C (793). The ternary troponin complex is a calcium-sensitive molecular apparatus that regulates the interaction of actin and myosin. Troponin T binds the troponin complex to tropomyosin, and troponin I binds to actin and inhibits interactions between actin and myosin. Troponin C is responsive to changes in intracellular calcium concentration. Amino acid sequences of the skeletal and cardiac isoforms of troponin I and troponin T have sufficient dissimilarity that monoclonal antibody-based immunoassays have been developed to detect cardiac-specific troponin T (cTnT) and cardiac-specific troponin I (cTnI). Because the amino acid sequence of troponin C is the same in cardiac and skeletal muscle, no immunoassays of troponin C have been developed for clinical purposes.

Because CK-MB is found in the skeletal muscle and blood of healthy subjects, the cutoff value for an elevated CK-MB level is typically set a few units above the upper end of the reference (normal) range. In contrast, because cardiac troponin I and cardiac troponin T are not normally detected in the blood of healthy people, the cutoff value for elevated cTnI and cTnT levels may be set only slightly above the noise level of the assay, permitting clinicians to diagnose lesser degrees of myocardial necrosis (ie, increased sensitivity) (796). Because CK and CK-MB are characteristically used as the gold standard for diagnosing MI, investigators may face a dilemma when a new diagnostic test is more sensitive than the gold standard, particularly for identifying episodes of minor myocardial cell necrosis. Case reports confirm histologic evidence of focal myocyte necrosis in patients with elevated cardiac troponin levels and normal CK values (796). It is estimated that ≈30% of patients presenting without ST-segment elevation who would otherwise be diagnosed with unstable angina are actually experiencing a non–Q-wave MI when assessed with cardiac-specific troponin assays (797). Furthermore, numerous investigators have now reported that elevated levels of cTnI or cTnT provide more prognostic information than that supplied by the patient's demographic characteristics or the ECG at presentation (798,799). Elevated cTnI or cTnT levels, even in the presence of normal CK-MB levels, identify patients without ST-segment elevation who are at an increased risk of death. Finally, patients presenting without ST-segment elevation who are characterized as high risk because of elevated cardiac-specific troponin levels demonstrate a greater benefit from treatment with new therapies such as glycoprotein (GP) IIb/IIIa inhibitors than patients without elevated cardiac-specific troponin levels who receive such new pharmacotherapeutic interventions (800).

CK-MB isoforms are another new serum cardiac marker that may be useful for evaluating patients with an acute coronary syndrome. CK-MB exists in only 1 form in myocardial tissue but in different isoforms (or subforms) in the plasma. An absolute level of CK-MB2 >1 U/L or a ratio of CK-MB2 to CK-MB1 of 1.5 has improved sensitivity and specificity for diagnosis of MI within the first 6 hours compared with conventional assays for CK-MB (59). Myoglobin, a low-molecular-weight heme protein found in cardiac and skeletal muscle, is not cardiac specific but is released more rapidly from infarcted myocardium than CK-MB and may be detected as early as 2 hours after MI. The diagnostic sensitivity and specificity for MI were compared for total CK-MB (activity and mass), CK-MB subforms, myoglobin, cTnI, and cTnT in the Diagnostic Marker Cooperative Study (DMCS) (801). The DMCS was a large, prospective, multicenter, double-blind study of patients presenting in the emergency department (ED) with chest pain. CK-MB subforms were most efficient for early diagnosis (within 6 h) of MI, whereas cTnI and cTnT were highly cardiac specific and particularly efficient for late diagnosis of MI. The DMCS investigators concluded that either a single assay (CK-MB subforms) or a select combination (CK-MB subform and a cardiac-specific troponin) reliably triages patients with chest pain and could potentially lead to improved therapy and reduced cost of care of ACS patients. It should be noted that serum levels of cTnT and cTnI may be present for several days after MI (up to 7 days for cTnI and up to 10 to 14 days for cTnT). Therefore, the ability to diagnose recurrent infarction is significantly compromised if the clinician relies solely on cardiac-specific troponins and fails to obtain a concomitant CK or CK-MB measurement within the first 12 to 24 hours of admission of an MI patient. Thus, although CK and CK-MB are not as cardiac specific as the troponins, they will return to normal levels within the first 24 to

36 hours, making it more likely that a reelevation is associated with recurrent myocardial necrosis. For patients presenting within the first two or three hours of symptom onset, the two markers most appropriate for the early diagnosis of AMI are myoglobin and CK-MB subforms.

In patients presenting with ST-segment elevation, clinicians usually use peak CK as a rough estimate of the magnitude of the infarct and assessment of the patient's prognosis. Release of cardiac-specific troponins is stoichiometrically correlated with the amount of myocardial necrosis, and the new serum cardiac markers can also be used to estimate infarct size and prognosis (58). Cardiac-specific troponins may not be detectable for up to 6 hours after onset of chest pain. Thus, when cTnI and cTnT levels are elevated early after onset of discomfort in patients with ST-segment elevation MI, clinicians should suspect that an antecedent episode of unstable angina was in fact MI and the patient is exhibiting a stuttering course of occlusion and release of the infarct-related artery. Data from the Global Utilization of Streptokinase and TPA for Occluded Arteries (GUSTO) III Study suggest that patients with elevated cardiac troponin T levels and who are <6 hours from the onset of discomfort have an increased mortality risk (802).

In addition to monitoring the patient for resolution of ischemic-type chest discomfort and regression of the magnitude of ST-segment elevation on the ECG, clinicians can obtain serial measurements of serum cardiac markers to buttress the noninvasive diagnosis of reperfusion of the infarct-related artery after thrombolytic therapy (65,803). Because of its rapid-release kinetics, myoglobin is a particularly attractive marker for the early diagnosis of reperfusion.

Bedside Testing for Serum Cardiac Markers

Handheld rapid bedside assays are clinically available for measuring cTnI, cTnT, myoglobin, and CK-MB. Small desktop rapid analyzers are also available for the same purpose. A rapid, high-voltage electrophoretic system is available for measuring CK-MB isoforms. When using a handheld rapid bedside assay for a serum cardiac marker, the clinician places a small aliquot of the patient's blood or serum in the specimen well and observes the development of a colored line in the read zone of the device. It should be noted that the time to development of the colored line and the intensity of the color are related to the concentration of the serum cardiac marker in the specimen. For example, when a handheld bedside immunoassay is used to test the blood of patients with high cTnT levels, a red line quickly appears; such patients are at increased mortality risk (804). Careful attention to the timing of the appearance of a positive bedside assay result may provide clinicians with a tool for a semiquantitative estimate of a serum cardiac marker level at the patient's bedside. A positive bedside test however should be confirmed by a conventional quantitative test.

Routine Measures (Oxygen, Nitroglycerin, Aspirin)

Recommendations

Class I

1. **Supplemental oxygen, intravenous access, and continuous electrocardiographic monitoring should be established in all patients with acute ischemic-type chest discomfort.**
2. **An ECG should be obtained and interpreted within 10 min of arrival in the ED in all patients with suspected acute ischemic-type chest discomfort.**

Although the specific diagnosis of acute MI can be made with absolute certainty only occasionally at the time of a patient's entry into the healthcare system, the immediate management of all acute coronary syndromes is generally the same. All patients suspected of having an acute MI should have a clinical and electrocardiographic evaluation that is prompt and targeted to estimate the likelihood that the presenting condition is an acute MI as opposed to one of its potentially lethal mimics: aortic dissection, acute pericarditis, acute myocarditis, spontaneous pneumothorax, or pulmonary embolism.

Although local settings vary widely, the entry process should be completed by a health team member (or members) with the competency to make such an assessment within a very short time of the patient's presentation, ideally within the first 10 minutes and certainly no more than 20 minutes from presentation. Only then should specific procedures or therapies be given, except for securing peripheral venous access. At this entry stage it is important that all members of the healthcare team interact with the patient and family in a warm and caring fashion while projecting professionalism and confidence.

Oxygen

Recommendations

Class I

1. **Overt pulmonary congestion.**
2. **Arterial oxygen desaturation (SaO_2) <90%.**

Class IIa

1. **Routine administration to all patients with uncomplicated MI during the first 2 to 3 h.**

Class IIb

1. **Routine administration of supplemental oxygen to patients with uncomplicated MI beyond 3 to 6 h.**

It has become universal practice to administer oxygen, usually by nasal prongs, to virtually all patients suspected of having acute ischemic-type chest discomfort, although it is not known whether this therapy limits myocardial damage or reduces morbidity or mortality. If oxygen saturation monitoring is used, therapy with supplemental oxygen is indicated if the saturation is less than 90%. Experimental results indicate that breathing oxygen may limit ischemic myocardial injury (66), and there is evidence oxygen administration reduces ST-segment elevation in patients with MI as well (67). The rationale for use of oxygen is based on the observation that even with uncomplicated MI, some patients are modestly hypoxemic initially, presumably because of ventilation-perfusion mismatch and excessive lung water (68).

In patients with severe CHF, pulmonary edema, or a mechanical complication of acute MI, significant hypoxemia may not be corrected with supplemental oxygen alone. Continuous positive-pressure breathing or endotracheal intubation and mechanical ventilation are often required in such cases and should not be unnecessarily delayed (69). A variety of mechanical ventilators are available, and multiple modes are possible. For patients who do

not have a depressed sensorium and are capable of initiating spontaneous ventilation, the preferred modes to use include intermittent mandatory ventilation, assist control, or pressure-support ventilation (70).

For patients without complications, it should be recalled that excess administration of oxygen can lead to systemic vasoconstriction, and high flow rates can be harmful to patients with chronic obstructive airway disease. On the other hand, because administration of nitroglycerin dilates the pulmonary vascular bed and increases ventilation-perfusion abnormalities, it is reasonable to provide supplemental oxygen, at least in the initial hours, for all patients suspected of having an acute MI. In the absence of compelling evidence for established benefit in uncomplicated cases and in view of its expense, there appears to be little justification for continuing its routine use beyond 2 to 3 hours.

Nitroglycerin

Recommendations for Intravenous Nitroglycerin

Class I

1. For the first 24 to 48 h in patients with acute MI and CHF, large anterior infarction, persistent ischemia, or hypertension.
2. Continued use (beyond 48 h) in patients with recurrent angina or persistent pulmonary congestion.

Class IIa

None.

Class IIb

1. For the first 24 to 48 h in all patients with acute MI who do not have hypotension, bradycardia, or tachycardia.
2. Continued use (beyond 48 h)* in patients with a large or complicated infarction.

Class III

1. Patients with systolic pressure <90 mm Hg or severe bradycardia (<50 bpm).

Considering that the use of nitrates in acute MI was believed to be contraindicated until the early 1970s (71), it is rather striking that today, with the exception of hypotensive patients, virtually all patients with acute ischemic syndromes will receive at least 1 sublingual nitroglycerin tablet before admission to the hospital. Aside from its known clinical benefit in alleviating ischemic myocardial pain, nitroglycerin is now appreciated as having a dilatory effect on the vascular smooth muscle in vessels throughout the body. Thus, vasodilation of the coronary arteries themselves (especially at or adjacent to sites of recent plaque disruption), the peripheral arteries, and the venous capacitance vessels is particularly beneficial to the patient with acute infarction. However, inadvertent systemic hypotension with resulting worsening of myocardial ischemia is the most serious potential complication of nitroglycerin therapy. Thus, patients with ischemic-type chest discomfort should receive sublingual nitroglycerin unless the initial systolic blood pressure is less than 90 mm Hg. It should be avoided in the presence of marked bradycardia (< 50 bpm) or tachycardia (72) and used with extreme caution, if at all, in patients with suspected right ventricular infarction. Patients with right ventricular infarction are especially dependent on adequate right ventricular preload to maintain cardiac output and can experience profound hypotension during administration of nitrates (73).

Long-acting oral nitrate preparations should be avoided in the early management of acute MI. Sublingual or transdermal nitroglycerin can be used, but intravenous infusion of nitroglycerin allows for more precise minute-to-minute control of this agent. Intravenous nitroglycerin can be successfully titrated by frequent measurement of cuff blood pressure and heart rate. Although invasive hemodynamic monitoring is not mandatory, it may be preferable if high doses of vasodilating agents are required, blood pressure instability ensues, or there is clinical doubt about the adequacy of LV filling pressure. Although quite effective in relieving ischemic-type chest discomfort due to acute coronary syndromes, nitroglycerin should not be used as a substitute for narcotic analgesia that is usually required to manage pain associated with acute MI. For a detailed discussion of the pharmacotherapy and relevant clinical studies pertaining to the use of nitroglycerin in acute MI, see Section V "Rationale and Approach to Pharmacotherapy."

Analgesia

The clinical observation of rapid and complete relief of pain after early reperfusion with thrombolytic therapy reinforces the concept that the pain of acute MI is due to continuing ischemia of viable but jeopardized myocardium rather than the effects of completed myocardial necrosis. Efforts to control pain therefore may reasonably involve use of anti-ischemic interventions, including, in addition to reperfusion, oxygen, nitrates, β-adrenoceptor blocking agents, and, in some circumstances, intra-aortic balloon counterpulsation. Effective analgesia (eg, intravenous morphine) should be administered promptly at the time of diagnosis and should not be delayed on the premise that to do so will obscure ability to evaluate the results of anti-ischemic therapy. See Section IV "Hospital Management" for more detailed discussion of proper analgesia.

Aspirin

Recommendations

Class I

1. A dose of 160 to 325 mg should be given on day 1 of acute MI and continued indefinitely on a daily basis thereafter.

Class IIb

1. Other antiplatelet agents such as dipyridamole, ticlopidine or clopidogrel may be substituted if true aspirin allergy is present or if the patient is unresponsive to aspirin.

The Second International Study of Infarct Survival (ISIS-2) has shown conclusively the efficacy of aspirin alone for treatment of evolving acute MI with a 35-day mortality reduction of 23% (29). When combined with streptokinase, the reduction in mortality was 42%. A meta-analysis demonstrated that aspirin reduced coronary reocclusion and recurrent ischemic events after thrombolytic therapy with either streptokinase or alteplase (74). In a dose of 160 mg or more, aspirin produces a rapid clinical antithrombotic effect caused by immediate and near-total inhibition of thromboxane A_2 production. Accordingly, aspirin now forms part of the early management of all patients with sus-

*Oral or topical preparations may be substituted

pected acute MI and should be given promptly and certainly within the first 24 hours at a dose between 160 and 325 mg and continued daily indefinitely.

Unlike fibrinolytic agents, there is little evidence for a time-dependent effect of aspirin on early mortality. However, data do support the contention that a chewable aspirin is absorbed more quickly than one swallowed in the early hours after infarction, particularly after opiate therapy. The use of aspirin is contraindicated in those with a hypersensitivity to salicylate and should be used with caution in patients with active ulcer disease. Aspirin suppositories (325 mg) can be used safely and are the recommended route of administration for patients with severe nausea and vomiting or known upper-gastrointestinal disorders. There is currently no evidence that other antiplatelet agents such as dipyridamole, ticlopidine, or sulfinpyrazone have any advantage over aspirin for mortality reduction after acute MI. See Section V "Rationale and Approach to Pharmacotherapy" for additional discussion on the use of aspirin in the management of acute MI, and Section VI "Preparation for Discharge From the Hospital."

Atropine

Recommendations

The following recommendations are applicable from early after onset of acute MI to 6 or 8 hours afterward:

Class I

1. **Sinus bradycardia with evidence of low cardiac output and peripheral hypoperfusion or frequent premature ventricular complexes at onset of symptoms of acute MI.**
2. **Acute inferior infarction with type I second- or third-degree atrioventricular (AV) block associated with symptoms of hypotension, ischemic discomfort, or ventricular arrhythmias.**
3. **Sustained bradycardia and hypotension after administration of nitroglycerin.**
4. **For nausea and vomiting associated with administration of morphine.**
5. **Ventricular asystole.**

Class IIa

1. **Symptomatic patients with inferior infarction and type I second- or third-degree heart block at the level of the AV node (ie, with narrow QRS complex or with known existing BBB).**

Class IIb

1. **Administration concomitant with (before or after) administration of morphine in the presence of sinus bradycardia.**
2. **Asymptomatic patients with inferior infarction and type I second-degree heart block or third-degree heart block at the level of the AV node.**
3. **Second- or third-degree AV block of uncertain mechanism when pacing is not available.**

Class III

1. **Sinus bradycardia >40 bpm without signs or symptoms of hypoperfusion or frequent premature ventricular contractions.**
2. **Type II AV block and third-degree AV block and third-degree AV block with new wide QRS complex presumed due to acute MI.**

By its parasympatholytic (anticholinergic) activity, atropine sulfate reduces vagal tone, enhances the rate of discharge of the sinus node, and facilitates AV conduction (75). It may be given as an adjunct to morphine administration when nausea and vomiting occur. During the early moments to hours of acute ischemia or acute MI, atropine is particularly useful in treating sinus bradycardia associated with reduced cardiac output and signs of peripheral hypoperfusion, including arterial hypotension, confusion, faintness, or frequent premature ventricular complexes (76). In this setting, leg elevation and intravenous administration of atropine may be lifesaving.

Atropine for Atrioventricular Block, Sinus Bradycardia, or Ventricular Asystole

Atropine is the drug of choice for the occasional treatment of type I second-degree AV block, especially when complicating inferior MI. It is occasionally useful in third-degree AV block at the AV node level in either restoring AV conduction or enhancing the junctional response. When AV block or sinus bradycardia is associated with CHF, hypotension, or frequent and complex ventricular arrhythmias, atropine may improve AV conduction, increase the sinus rate, and avoid the need for immediate insertion of a transvenous pacemaker (77). As a rule, however, in the absence of hemodynamic compromise, treatment of sinus bradycardia or first- or second-degree AV block is not indicated. Similarly, atropine is rarely, if ever, the drug of choice for management of type II second-degree AV block. On occasion, while failing to improve AV block, atropine may increase the sinus rate, and, in fact, enhance the block.

The recommended dosage of atropine for bradycardia is 0.5 to 1.0 mg intravenously (IV), repeated if needed every 3 to 5 minutes to a total dose of no more than 2.5 mg (0.03 to 0.04 mg/kg), the amount that produces complete vagal blockade. Atropine may also be therapeutic in ventricular asystole, for which the recommended dose is 1 mg IV, to be repeated every 3 to 5 minutes (while CPR continues) if asystole persists. The total cumulative dose should not exceed 2.5 mg over 2.5 hours. The peak action of atropine given intravenously is observed within 3 minutes (1).

Side Effects

When administered in doses of less than 0.5 mg or other than intravenously, atropine may produce a paradoxic effect (namely, bradycardia and depression of AV conduction) (78), which is due either to central reflex stimulation of the vagus or a peripheral parasympathomimetic effect on the heart. Urinary retention is not uncommon following administration of atropine and can be deleterious to the patient with acute MI. Repeated administration of atropine may produce adverse central nervous system effects, including hallucinations and fever. Careful dosing and observation after administration of atropine is necessary because the sinus tachycardia that follows may increase ischemia. Rarely, ventricular tachycardia and fibrillation occur after intravenous administration of atropine (79).

Pacing is the treatment of choice for symptomatic bradycardia

not responding promptly to atropine administration.

Risk Stratification and Management of ST-Segment Elevation/Bundle Branch Block Cohort

Newer Concepts

The spectrum of myocardial ischemia consists of patients with clinical presentations that cover the following range of diagnoses: stable angina, unstable angina, MI without ST elevation, and MI with ST elevation. Clinical discrimination among unstable angina, Q wave, and non–Q-wave MI can only be made retrospectively after serial ECGs and serum cardiac markers have been obtained (Figure 2). Patients with ST-segment elevation have a high likelihood of a coronary thrombus occluding the infarct-related artery (80,81). However, not every ST-elevation MI evolves into a Q-wave MI. Angiographic evidence of occlusive coronary thrombus formation may be seen in more than 90% of patients with ST-elevation MI but in only 1% of patients with stable angina and about 35% to 75% of patients with unstable angina or non–Q-wave MI (80-83). Commonly indicated treatment regimens for all acute coronary ischemic syndromes include aspirin, heparin, β-adrenoceptor blockers, and nitrates. Thrombolytic therapy is highly effective in patients with ST elevation or presumably new LBBB (which obscures the electrocardiographic diagnosis of MI)(27) (Figure 3). At the same time, evidence now suggests that thrombolytic therapy is ineffective (for normal or nonspecific electrocardiographic presentations) and possibly even harmful (for ST-depression presentation) in unstable angina and non–ST-elevation MI subgroups (27,84). Figure 4 presents a suggested schema for management of acute MI without ST-segment elevation.

Noninvasive Imaging in the Emergency Department

Screening patients who present with ischemic-type chest discomfort in the ED is an area of clinical and economic importance. Because only 25% or less of patients admitted to the hospital to "rule out" MI actually suffer an MI, accurate screening techniques to identify patients with ongoing necrosis is an important goal. The usefulness of echocardiography in the ED as a means of screening for MI has been validated, but small areas of infarction can be missed, and the age of a regional wall motion abnormality cannot be determined (85-87a). Thallium and sestamibi imaging in the ED are both very good radioisotope screening techniques (85,88,89) and appear to be quite sensitive. However, their use in the ED is still viewed as experimental and is not recommended. In time, the value of noninvasive imaging may further diminish as rapid assays of specific, earlier, and more sensitive serum markers of myocardial necrosis are developed (56,58,60,61).

Thrombolysis

Recommendations

The constellation of clinical features that must be present (although not necessarily at the same time) to serve as standard indications for administration of thrombolytic therapy to patients with acute MI are as follows: (selection of specific thrombolytic agents or regimens is discussed in "Rationale and Approach to Pharmacotherapy.")

Class I

1. **ST elevation (>0.1 mV, two or more contiguous leads),* time to therapy 12 h or less,† age <75 years.**
2. **Bundle branch block (obscuring ST-segment analysis) and history suggesting acute MI.**

Comment: Treatment benefit is present regardless of gender, presence of diabetes, blood pressure (if < 180 mm Hg systolic), heart rate, or history of previous MI (27). Benefit is greater in the setting of anterior MI, diabetes, low blood pressure (< 100 mm Hg systolic), or high heart rate (> 100 bpm). The earlier therapy begins, the better the outcome, with the greatest benefit decidedly occurring when therapy is given within the first 3 h; proven benefit occurs, however, up to at least within 12 h of the onset of symptoms. Benefit is less with inferior acute MI, except for the subgroup with associated right ventricular infarction (ST elevation RV-4) or anterior-segment depression indicative of a posterior current of injury as often occurs with occlusion of a large circumflex coronary artery.

Class IIa

1. **ST elevation,* age 75 years or older.**

Comment: In persons older than 75 years, the overall risk of mortality from infarction is high without and with therapy. Although the proportionate reduction in mortality is less than in patients younger than 75, the absolute reduction results in 10 lives saved per 1000 patients treated in those over 75. The relative benefit of therapy is reduced (27).

Class IIb

1. **ST elevation,* time to therapy > 12 to 24 h,†**
2. **Blood pressure on presentation >180 mm Hg systolic and/or >110 mm Hg diastolic associated with high-risk MI.**

Comment: Generally there is only a small trend for benefit of therapy after a delay of >12 to 24 h, but thrombolysis may be considered for selected patients with ongoing ischemic pain and extensive ST elevation. Risk of intracranial hemorrhage (ICH) is greater when presenting blood pressure is greater than 180/110 mm Hg, and in this situation the potential benefit of therapy must be weighed carefully against the risk of hemorrhagic stroke. Risk of cardiac rupture appeared to increase with prolonged time to therapy in an earlier meta-analysis (90) but was not associated with increased risk of rupture in a later, larger study (91). Generally patients presenting >12 h after symptom onset were excluded from some but not all trials. An attempt to lower blood pressure first (with nitrates, β-adrenoceptor blockers, etc) is recommended but is not of proven benefit in lowering the risk of ICH. Primary PTCA or CABG may be considered if available.

Class III

1. **ST elevation,* time to therapy >24 h,† ischemic pain resolved.**

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* Repeat ECGs recommended during medical observation in suggestive clinical settings when initial ECG is nondiagnostic of ST elevation.

† Time of symptom onset is defined as the beginning of continuous, persistent discomfort that brought the patient to the hospital.

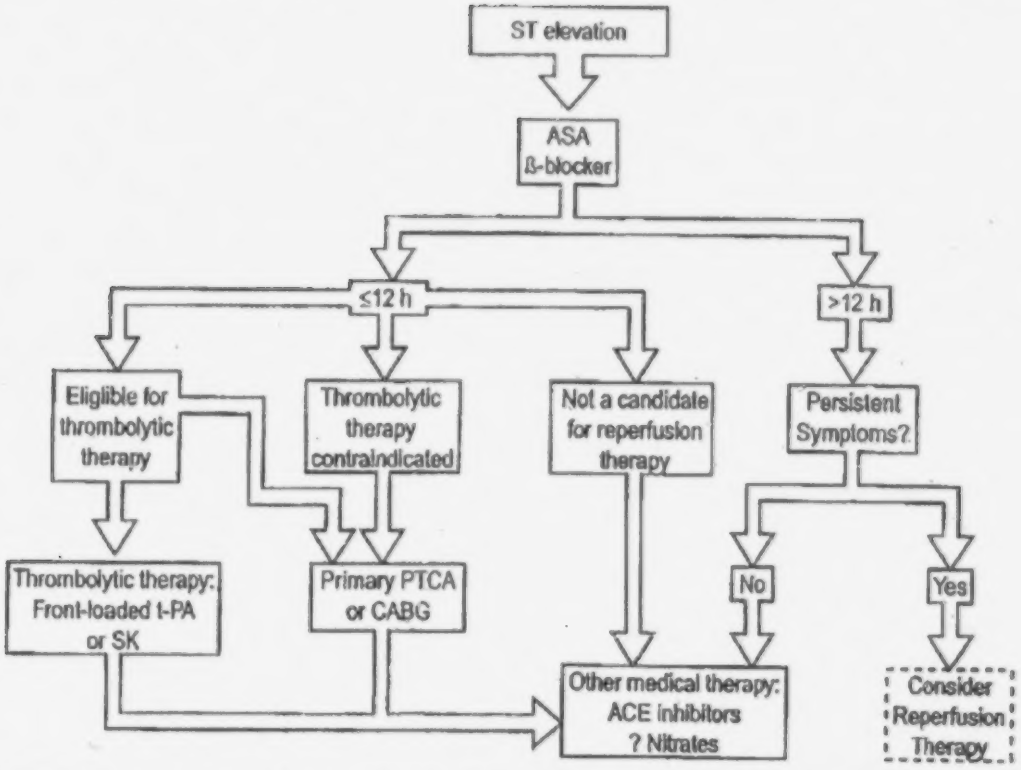

Figure 3. Recommendations for management of patients with ST elevation. All patients with ST-segment elevation on the electrocardiogram should receive aspirin (ASA), ß-adrenoceptor blockers (in the absence of contraindications), and an antithrombin (particularly if tissue-type plasminogen activator [tPA] is used for thrombolytic therapy). Whether heparin is required in patients receiving streptokinase (SK) remains a matter of controversy; the small additional risk for intracranial hemorrhage may not be offset by the survival benefit afforded by adding heparin to SK therapy. Patients treated within 12 hours who are eligible for thrombolytics should expeditiously receive either front-loaded tPA or SK or be considered for primary percutaneous transluminal coronary angioplasty (PTCA). Primary PTCA is also to be considered when thrombolytic therapy is absolutely contraindicated. Coronary artery bypass graft (CABG) may be considered if the patient is less than 6 hours from onset of symptoms. Individuals treated after 12 hours should receive the initial medical therapy noted above and, on an individual basis, may be candidates for reperfusion therapy or angiotensin-converting enzyme (ACE) inhibitors (particularly if left ventricular function is impaired). Modified from Antman EM. Medical therapy for acute coronary syndromes: an overview. In Califf RM, ed. Atlas of Heart Diseases, VIII. Philadelphia, Pa: Current Medicine, 1996.

2. ST-segment depression only.

Comment: In the absence of ST elevation, there is no evidence of benefit for patients with normal electrocardiographic or nonspecific changes, and, using current thrombolytic regimens, there is some suggestion of harm (including increased bleeding risk) for patients with ST-segment depression only (27,92). When marked ST-segment depression is confined to leads V_1 through V4, there is a likelihood that this reflects a posterior current of injury and suggests a circumflex artery occlusion for which thrombolytic therapy would be considered appropriate. Very recent retrospective analysis of the Late Assessment of Thrombolytic Efficacy (LATE) Trial (93,94) also casts some uncertainties about withholding thrombolytic therapy from this heterogeneous group of patients.

A collaborative overview from nine trials of thrombolytic therapy (vs. control) for acute MI has shown a highly significant ($P<0.00001$) 18% proportional reduction in 35-day mortality (9.6% fibrinolysis vs. 11.5% control) corresponding to a reduction of 18 deaths per 1000 patients treated when data from all patient groups are pooled (27). In patients with ST elevation, a proportional mortality reduction of 21% occurred. It is now known that this survival benefit can be maintained long term (6 months to at least 4 years) (27,95).

Figure 5 summarizes the number of lives saved per 1000 patients treated based on the presenting ECG pattern (96). In general, thrombolytic agents should be administered only to patients with ST-segment elevation greater than 0.1 mV or presumably new LBBB on the ECG (27,97). However, in the very early phase of acute infarction, giant, hyperacute T waves may be present with no ST-segment elevation. Similarly, direct posterior infarction can result in ST-segment depressions in leads V_1 through V_4, and in both situations it is appropriate to administer thrombolytic therapy. Thus, it should be clear that certain cases require experienced interpretation of the ECG before withholding reperfusion therapy. Unquestionably, patients with LBBB and anterior ST elevation are at greater inherent risk from MI but also achieve greater benefit with thrombolytic therapy. Although one study (39) suggested that the amount of ST elevation might also predict greater inherent risk and therefore greater benefit, it did not take into account

New Table 2.1 Intracranial Hemorrhage in Recent Thrombolytic Trials

Patient Characteristics	GUSTO-I (497)	GUSTO-II (805)	GUSTO-III (802, 833)	COBALT (832)	ASSENT-2*	In Time-II*
Number	41,021	3473	15,059	7169	16,950	15,078
Average Age (y)	62	62.5	63	62.4	–	–
>75 y (%)	10.5	11.8	13.6	13.0	–	–
Female (%)	25.2	22.4	27.4	23.4	–	–
Intracranial Hemorrhage Rates						
SK	0.51	0.37	–	–	–	–
tPA	0.70	0.72	0.87	Double bolus 1.12 Accl infusion 0.81	0.93	0.62
rPA	–	–	0.91	–	–	–
TNK-tPA	–	–	–	–	0.94	–
nPA	–	–	–	–	–	1.13

accl indicates accelerated; nPA, lanoteplase; rPA, reteplase; TNK-tPA, a genetically engineered variant of tPA; tPA, tissue plasminogen activator; SK, streptokinase.
*Data based on preliminary results

increasing absolute mortality rates with advancing age result in progressively greater absolute mortality reductions up to age 75. Benefit may also be achieved after age 75 but is less certain than at younger ages (27,101). Advanced age does increase risk of stroke after acute MI, both without and with thrombolytic therapy. Given the much greater mortality risk of MI, the elderly should be considered candidates for thrombolytic therapy after careful screening for exclusions. Patients should be considered at higher risk if they have any of the following: female gender, advanced age (>70 years), history of previous infarction, atrial fibrillation, anterior infarction, rales in more than one third of the lung fields, hypotension, and sinus tachycardia or diabetes mellitus (27,102). Indeed, certain subgroups of patients with an especially high likelihood of benefiting from successful reperfusion include those with hypotension, tachycardia, and a history of diabetes mellitus or prior MI.

Early placebo-controlled trials of thrombolysis for MI raised concern about a paradoxical increase in mortality during the first 24 hours after thrombolysis that was later offset by a greater reduction in mortality in the thrombolytic groups (27). More recently conducted thrombolytic trials have confirmed a "high density" of mortality in the first 24 hours but suggest that this may be attributed primarily to pump failure from unsuccessful reperfusion rather than an early hazard of thrombolysis (103).

Risk of Stroke

Thrombolytic therapy is associated with a slight but definite excess risk of stroke that occurs predominantly within the first day of therapy (27). Clinical variables that can be ascertained in the ED that predict an increased risk of ICH are advanced age (older than 65 years, odds ratio 2.2, 95% CI, 1.4 to 3.5), low body weight (< 70 kg, odds ratio 2.1, CI 1.3 to 3.2), hypertension on presentation (odds ratio 2.0, CI 1.2 to 3.2), and use of alteplase (odds ratio 1.6, CI 1.0 to 2.5) (104-106). The number of risk factors at presentation may be used to estimate the probability of ICH and is shown in Figure 8*. Although no firm guidelines have been established, ICH rates less than 1% have generally been regarded as acceptable in clinical trials, considering the overall favorable benefit-risk profiles, whereas rates greater than 1.5% have been viewed as unacceptably high (107).

* To reduce the risk, the dose of 90-minute alteplase should be adjusted downward for low body weight (<67 kg). Similarly the 180-minute regimen should be adjusted downward for patients who weigh <65 kg.

More recent trials show that as use of thrombolysis has increased, a greater proportion of patients who are >75 years old or female are now included. This change has been associated with a higher rate of ICH than that seen in earlier studies. For example, the rate of ICH after administration of alteplase was ≈0.7%; in more recent studies, it is 0.8% to 0.9% (New Table 2.1). It should be noted that the streptokinase without heparin administration regimen has the lowest rate of ICH.

Net Clinical Benefit

Clinicians must carefully weigh the risk-benefit ratio of thrombolysis for individual patients. Hesitancy to prescribe thrombolytic therapy arises from concern about intracranial bleeding and uncertainty about eligibility criteria. The generally higher mortality rate among MI patients who do not undergo thrombolysis underscores the need for heightened awareness of current indications for thrombolysis through such projects as the NHAAP (108). Decision analysis methods suggest that appropriate use of thrombolytic therapy in eligible patients would save many additional lives annually in the United States (109).

Contraindications/Cautions

Hemorrhage represents the most important risk of thrombolytic therapy, especially ICH, which may be fatal in one half to two thirds of patients. Contraindications and cautions to thrombolytic use are given in Table 3.

Summary of Initial Diagnostic and Treatment Strategy

A summary of initial diagnostic and treatment strategies for patients with acute MI with ST elevation or BBB, focusing on emergency management, is provided in Table 4.

Primary Percutaneous Transluminal Coronary Angioplasty

Recommendations

Class I

1. **As an alternative to thrombolytic therapy in patients with AMI and ST-segment elevation or new or presumed new LBBB who can undergo angioplasty of the infarct-related artery within 12 h of onset of symptoms or >12 h if ischemic symptoms persist, if performed in a timely**

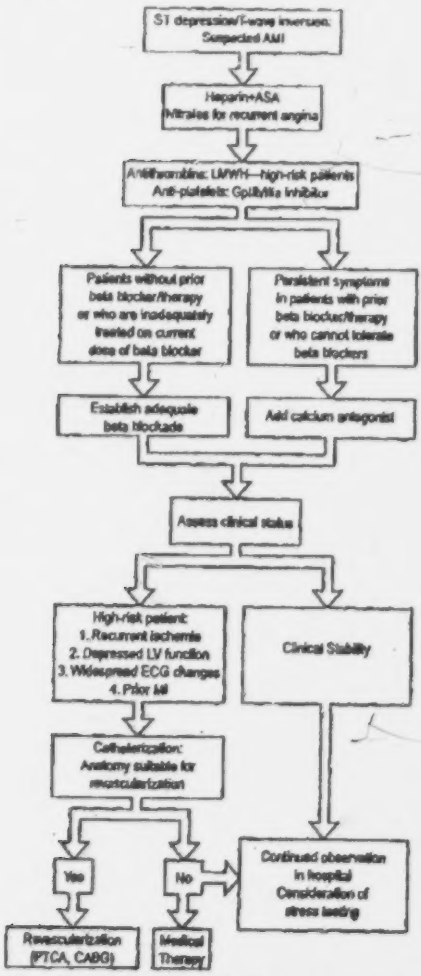

Figure 4. All patients *without* ST elevation should be treated with an antithrombin and aspirin (ASA). Nitrates should be administered for recurrent episodes of angina. Adequate β-adrenoceptor blockade should then be established; when this is not possible or contraindications exist, a calcium antagonist can be considered. Current data indicate that either an invasive or non-invasive treatment strategy is suitable for non-ST-elevation AMI patients. AMI indicates acute myocardial infarction; CABG, coronary artery bypass graft; ECG, electrocardiogram; GpIIb/IIIa, Glycoprotein IIb/IIIa receptor for platelet aggregation; LMWH, low-molecular-weight heparin; LV, left ventricular; PTCA, percutaneous transluminal coronary angioplasty. Modified from Asensio EM. Medical therapy for acute coronary syndromes: an overview. In Califf RM, editor. Atlas of Heart Diseases, VIII. Philadelphia, PA: Current Medicine; 1996.

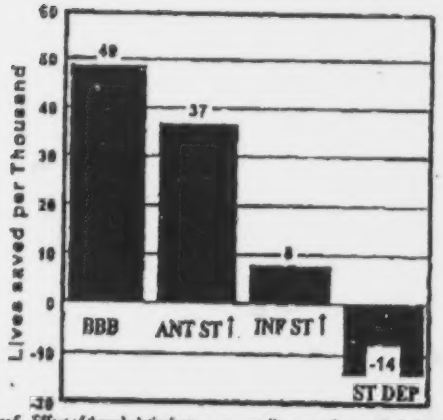

Figure 5. Effect of thrombolytic therapy on mortality according to admission electrocardiogram. Patients with bundle branch block (BBB) and anterior ST-segment elevation (ANT ST↑) derive the most benefit from thrombolytic therapy. Effects in patients with inferior ST-segment elevation (INF ST↑) are much less, while patients with ST-segment depression (ST DEP) do not benefit. Adapted from Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomized trials of more than 1000 patients. Lancet Ltd. 1994;343:311-322. Reprinted from Management of Acute Myocardial Infarction (Julian D, Braunwald E, eds). Martin GV, Kennedy JW. Choice of thrombolytic agent, p 90, 1994, by permission of the publisher, WB Saunders Co Ltd, London.

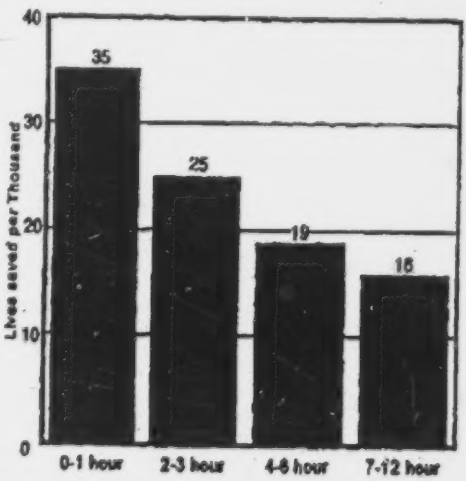

Figure 6. Effect of thrombolytic therapy on mortality according to time from symptom onset. Patients treated early derive the most benefit. Adapted from Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomized trials of more than 1000 patients. Lancet Ltd. 1994;343:311-322. Reprinted from Management of Acute Myocardial Infarction (Julian D, Braunwald E, eds). Martin GV, Kennedy JW. Choice of thrombolytic agent, p 90, 1994, by permission of the publisher, WB Saunders Co Ltd, London.

the increased amount of ST elevation seen in patients with anterior infarction. Other factors such as collateral flow (98) clearly influence the amount of ST elevation, which may limit its value for predicting therapeutic benefit.

Additional factors that influence the decision to administer thrombolytic therapy include time since onset of symptoms, patient's age, hemodynamic status, and coexisting medical illnesses (Figure 6 and Figure 7). Myocardial salvage increases with progressively earlier administration of thrombolytic therapy, although a reduction in mortality may still be seen in patients treated up to at least 12 hours from onset of definitive symptoms (27,99,100). Some patients presenting at more than 12 to 24 hours with persistent ischemic symptoms and ST elevation also may benefit from treatment. Although younger patients achieve a greater relative reduction in mortality compared with older patients, the

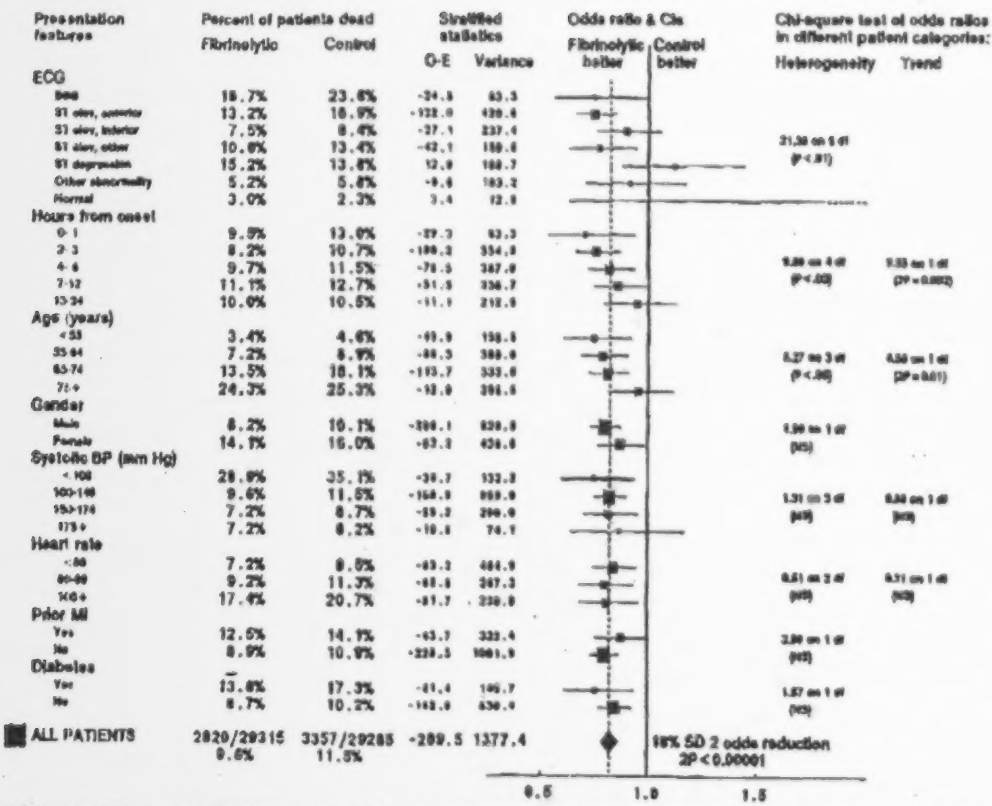

Figure 7. Mortality differences during days 0 through 35 subdivided by presentation features in a collaborative overview of results from nine trials of thrombolytic therapy. At center absolute mortality rates are shown for fibrinolytic and control groups for each clinical feature at presentation listed at left. The odds ratio of death in fibrinolytic group to that in control group is shown for each subdivision (black square) along with 95% confidence interval (horizontal line). The summary odds ratio at bottom corresponds to an 18% proportional reduction in 35-day mortality and is highly statistically significant. This translates to a reduction of 18 deaths per 1000 patients treated with thrombolytic agents. O-E indicates observed versus expected ratio; CIs, confidence intervals; ECG, electrocardiogram; BBB, bundle branch block; ST elev, ST-segment elevation; df, degrees of freedom; BP, blood pressure; MI, myocardial infarction; SD, standard deviation. Adapted from Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomised trials of more than 1000 patients. Lancet Ltd. 1994;343:311-322. Reprinted from Management of Acute Myocardial Infarction (Julian D, Braunwald E, eds). Martin GV, Kennedy JW. Choice of thrombolytic agent, p 42-44 , 1994, by permission of the publisher, WB Saunders Co Ltd, London.

fashion* by persons skilled in the procedure† and supported by experienced personnel in an appropriate laboratory environment.‡

2. In patients who are within 36 h of an acute ST-elevation/Q-wave or new LBBB MI who develop cardiogenic shock, are <75 years of age, and revascularization can be performed within 18 h of onset of shock.

Class IIa

1. As a reperfusion strategy in candidates for reperfusion who have a contraindication to thrombolytic therapy (Table 3).

Class IIb

1. In patients with AMI who do not present with ST elevation but who have reduced (less than TIMI [Thrombolysis in Myocardial Infarction] grade 2) flow of the infarct-related artery and when angioplasty can be performed within 12 h of onset of symptoms.

Class III

This classification applies to patients with AMI who:

1. Undergo elective angioplasty of a non–infarct-related artery at the time of AMI
2. Are >12 h after onset of symptoms and have no evidence of myocardial ischemia
3. Have received fibrinolytic therapy and have no symptoms of myocardial ischemia
4. Are eligible for thrombolysis and are undergoing primary angioplasty performed by a low volume operator in a laboratory without surgical capability

Comment: There is serious concern that a routine policy of primary PTCA for patients with acute MI will result in unacceptable delays in achieving reperfusion in a substantial number of cases and less than optimal outcomes if performed by less experienced operators. Strict performance criteria must be mandated for primary angioplasty programs so that

* Performance standard: balloon inflation within 90±30 minutes of admission.
‡ Individuals who perform >75 PTCA procedures per year (110).
† Centers that perform >200 PTCA procedures per year and have cardiac surgical capability (110).

Incidence	53%	31%	15%	1%
Likelihood Ratio	0.34	1.28	1.77	2.89

Figure 8. Risk of intracranial hemorrhage (ICH) during thrombolytic therapy. At bottom is estimated incidence of frequency of one or more of the following risk factors: age >65 years, weight <70 kg, hypertension on admission, and use of tissue plasminogen activator (TPA) in patients with acute MI who are potential candidates for thrombolytic therapy. The likelihood ratio describes the probability of finding the risk profile among patients with intracranial bleeding divided by the probability of finding the same risk profile among patients without intracranial bleeding. Curves depict estimated probability of ICH assuming an overall incidence of 0.5% and 0.75% (bottom and top curves respectively). Adapted from data in Simoons ML, MAggioni AP, Knatterud G, et al. Individual risk assessment for intracranial hemorrhage during thrombolytic therapy. Lancet Lal. 1993;342:1523-1528

Table 3. Contraindications and Cautions for Thrombolytic Use in Myocardial Infarction*

Contraindications
- Previous hemorrhagic stroke at any time; other strokes or cerebrovascular events within 1 year
- Known intracranial neoplasm
- Active internal bleeding (does not include menses)
- Suspected aortic dissection

Cautions/relative contraindications
- Severe uncontrolled hypertension on presentation (blood pressure >180/110 mm Hg)†
- History of prior cerebrovascular accident or known intracerebral pathology not covered in contraindications
- Current use of anticoagulants in therapeutic doses (INR ≥2-3); known bleeding diathesis
- Recent trauma (within 2-4 weeks), including head trauma or traumatic or prolonged (>10 min) CPR or major surgery (<3 wk)
- Non compressible vascular punctures
- Recent (within 2-4 weeks) internal bleeding
- For streptokinase/anistreplase: prior exposure (especially within 5 days-2 y) or prior allergic reaction
- Pregnancy
- Active peptic ulcer
- History of chronic severe hypertension

INR indicates International Normalized Ratio; CPR, cardiopulmonary resuscitation.
*Viewed as advisory for clinical decision making and may not be all-inclusive or definitive.
†Could be an absolute contraindication in low-risk patients with myocardial infarction (see text).

such delays in revascularization and performance by low-volume operators/centers do not occur. Interventional cardiologists and centers must operate within a specified "corridor of outcomes" to include (1) balloon dilation within 90±30 min of admission and diagnosis of AMI; (2) a documented clinical success rate with Thrombolysis in Myocardial Infarction (TIMI) II through III flow attained in >90% of patients without emergency CABG, stroke, or death; (3) emergency CABG rate <3% among all patients undergoing the procedure; (4) actual performance of angioplasty in a high percentage of patients (85%) brought to the laboratory; and (5) mortality rate <10%. Otherwise, the focus of treatment should be the early use of thrombolytic therapy.

Table 4. Diagnostic and Treatment Measures in Patients With ST Elevation or Bundle Branch Block

Initial diagnostic measures
1. Use continuous ECG, automated BP, HR monitoring
2. Take targeted history (for AMI inclusions, thrombolysis exclusions), check vital signs, perform focused examination
3. Start IV(s), draw blood for serum cardiac markers, hematology, chemistry, lipid profile
4. Obtain 12-lead ECG
5. Obtain chest x-ray (preferably upright)

General treatment measures
1. Aspirin, 160-325 mg (chew and swallow)
2. Nitroglycerin, sublingual: test for Prinzmetal's angina, reversible spasm; anti-ischemic, antihypertensive effects
3. Oxygen: sparse data; probably indicated, first 2-3 h in all; continue if low arterial oxygen saturation (<90%)
4. Adequate analgesia: small doses of morphine (2-4 mg) as needed

Specific treatment measures
1. Reperfusion therapy: goal—door-to-needle time <30 min; door-to-dilation time <60 min
2. Conjunctive antithrombotics: aspirin, heparin (especially with TPA)
3. Adjunctive therapies: β-adrenoceptor blockade if eligible, intravenous nitroglycerin (for anti-ischemic or antihypertensive effects), ACE inhibitor (especially with large or anterior AMI, heart failure without hypotension [SBP >100 mm Hg], previous MI).

ECG indicates electrocardiogram; BP, blood pressure; HR, heart rate; AMI, acute myocardial infarction; IVs, intravenous administrations; TPA, tissue plasminogen activator; ACE, angiotensin converting enzyme; SBP, systolic blood pressure.

Since publication of the original report of primary (direct) PTCA as an alternative to thrombolytic therapy in patients with acute MI, (111) its merits have been debated (112,113). There are no randomized controlled trials of primary PTCA versus no reperfusion. Thus, the recommendations are based on findings from small and moderately sized comparative trials of primary PTCA and thrombolysis and from indirect evidence.

Initial assessments showed that PTCA restored antegrade flow in the occluded infarct-related artery in more than 90% of patients and was associated with a 1-year survival rate of 90% to 96% (114-117). Subsequently several randomized trials compared PTCA and thrombolytic therapy in patients with acute MI (118-120). In these studies PTCA was reported to successfully restore antegrade coronary flow in approximately 88% to 95% of attempts. In the study of Zijlstra et al, (118) follow-up angiography weeks after infarction showed that the infarct-related artery was patent in 91% of those who had primary PTCA (studied on average 3 months after the procedure) and in 68% of those who received streptokinase (P=.001) (studied 3 weeks later), and the residual infarct-related artery stenosis was less in those who underwent PTCA. Those in whom primary PTCA was performed also had fewer in-hospital adverse events (nonfatal reinfarction or death) and were less likely to have recurrent ischemia or to require coronary revascularization over the period of follow-up.

Similarly, Gibbons et al (119) found that those who underwent

APPENDIX H

[Page 10] hate to ask you to read back but—

THE WITNESS: I'll bring a copy of my resume.

MR. BEASLEY: No, no. She'll just tell me what it is. What was his answer?

(The requested portion of the record was read by the reporter.)

Q. (BY MR. ROGERS) Next question. You position at Baptist Memorial Hospital, could you repeat what your position is?

A. I'm the medical director.

Q. Dr. Andershock, what were your work hours on October 6, 2000? October 6, 2000—

A. Was the date we saw her—okay—Ms. Dulan.

Q. Yes.

A. I was working the day shift; 7 a.m. to 4 p.m.

Q. Did you stay any longer than four o'clock?

A. Yes.

Q. Was there any particular reason that you stayed over?

A. Yeah. There were a lot of patients.

[Page 11] It was very busy, and I had to stay over to get patients taken care of—disposition.

Q. Were you the only ER doctor at the time that Plaintiff came in the emergency room?

A. I see. We have—I was on the trauma end. There was another physician in the middle. I think we had three physicians working at the time, including myself.

Q. Do you know what time Plaintiff came to—when I refer to Plaintiff, that's Jeannette Rogers Duan.

A. Yes, sir.

Q. Do you know what time Plaintiff came to the Huntsville Hospital's emergency room?

A. The record shows that she signed in at 1302.

Q. Plaintiffs deposition that the hospital did of Plaintiff a little while back disputes that time. I'm just pointing it out. At page 78 of the Plaintiffs deposition, Plaintiff maintains that she came in at 12:30. Her mother, who was also deposed by the hospital; states at page 33 of her deposition—if anyone wants to review

[Page 12] any of these at any time,. we can—that it was sometime after 12 noon. I'm just putting that in the record so that if there's some dispute as to the time frames.

Approximately what time did you become involved in Plaintiffs care and treatment?

MR. BEASLEY: I object to the form of the question. Move to strike statements on the record if they're intended to be a part of the question. Go ahead.

A. The record shows that I saw her at 1440.

Q. How long was Plaintiff under your . care?

A. Plaintiff was—Ms. Rogers Dulan was under my care from I440 to approximately 1715.

Q. Okay. Could you convert those numbers?

A. I'm sorry. I don't understand. Convert which way?

MR. BEASLEY: Not military.

A. Oh, I'm sorry. From 2:40 to 5:15.

Q. Okay. Dr. Andershock, I'm just going

[Page 13] to point out just a discrepancy in the time recollection based upon the hospital's deposition of Plaintiff as well as Plaintiffs mother. Plaintiff states in her deposition at pages 155 through 156 that she saw you at approximately 3:00 —somewhere between 3:00 to 3:45.

MR. BEASLEY: Until he puts a question mark on it, Dr. Andershock, don't nod or don't respond. When he puts a question mark on this, then we'll deal with it. But I can't object until he puts a question mark on it. The statements that end in a period are apparently for the record, but let's don't respond until we have a question mark.

THE WITNESS: Okay.

Q. (BY MR. ROGERS) Mildred Rogers, who was deposed by the hospital, states in her deposition at pages 59 and 66 that she remembers looking at her watch and that you came to interview her at approximately—that is, the Plaintiff; sometime after 3:00. I'll just repeat for—the hospital's attorney, Mr. Beasley, during that deposition

[Page 38] A. No. I'm trying to phrase my answer. The answer is it depends. You can have different presentations for a heart attack. I've had patients who are in obvious excruciating pain and discomfort, and I've had patients who are having a heart attack that had no symptoms at all. So what are best questions? The ones that we ask all the time are, you know,. chest pain, shortness of breath, nausea, vomiting, sweating. But my personal experience has been that even in asking those questions, that is not going to be enough to determine what's going on.

Q. Well, would you say that Plaintiff was exhibiting those symptoms? Sweating—

A. What she had told me was that she was having chest pain that was radiating to her arm and to her back, and she was nauseous, had vomited and was short of breath, and then when it started, she had gotten sweaty.

Q. Dr. Andershock, would the added fact that Plaintiff was 15 years. postmenopausal, would that have increased the risk that maybe this was a heart attack or that Plaintiff had a history of hypertension?

[Page 39] A. The fact that she was postmenopausal and her history of hypertension—

Q. Fifteen years postmenopausal..

A. —represents supporting evidence. The clinical presentation at this point was strong enough that you didn't need that to move forward.

Q. Dr. Andershock, what is the profile of persons who are at greatest risk for a heart attack? How would you describe that just to the best of your ability?

A. I'm trying to remember from the AHA guidelines. Certainly diabetes is a significant risk factor, high blood pressure, women being postmenopausal or certainly being, over the age of 40 years in association of hypertension, high cholesterol and a family history of coronary artery disease.

MR. BEASLEY: Smoking? Does that count?

Q. (BY MR. ROGERS) So Plaintiff—in the records that were in the hospital, if we can go through them, Plaintiff was fifty-five years old so that issue—was 15 years postmenopausal. So you would say that those [Page 40] were risk factors, correct?

A. Yes.

Q. The records indicate—and you can dispute this if you wish. The records indicate that Plaintiffs family medical history listed myocardial infarction. Risk factor?

A. The family history?

Q. Right.

A. Yes. Well, actually was the onset after the age of fifty-five or before the age of fifty-five? There is actually an added level where if the onset of coronary artery disease in a parent is after the age of fifty-five, although it is still relevant, it is not as pertinent.

Q. Okay. What about the records that indicated that Plaintiff was experiencing substantial chest pain that radiates to her neck and shoulder and shortness of breath? Would that be considered to be an. indication that maybe Plaintiff was possibly suffering from a heart attack?

A. Her clinical presentation was consistent with anterior--or with, I'm [Page 40 sorry, cardiac ischemia, yes.

Q. Dr. Andershock, one question that comes up frequently whenever I'm discussing this case with anyone is with respect to your—I've been told that these are called T sheets.

A. Right.

Q. All right. With respect to the severity of pain, I think you have three choices here. Mild, moderate, severe. And you circled mild. I've just—I've always wanted to ask you that.

MR. BEASLEY: Ask what?

Q. (BY MR. ROGERS) I want to ask Dr. Andershock why he circled Plaintiffs pain as being mild?

MR. BEASLEY: Object to the form.

A. Again, trying to remember, you know, two and a half years ago the specific instances in the conversation that we had is difficult. These are questions that you ask the patient directly, and I usually write down what they say.

Q. Okay.

[Page 82] try and get it wherever they can, and that's their job. So if they see an opportunity to get that information, they will go, you know, and get that.

Now, that information is important to us because we use that to register them to get them into the computer system to order tests. So it's important that that job be done, and that is their only job to get these people registered into the computer system.

And, again, I'm reading into this. I don't know what happened, and personally, I think if we could talk to the people who were involved, they might be able to be more clear. But perhaps that person saw an opportunity to get this information from Dr. Dulan so she could. be registered into the computer and entered into the computer so we could order whatever test or whatever therapies that needed to be done.

Q. Okay; Plaintiff ultimately was taken back to a small treatment room, and there's just time discrepancies there with respect to how long she stayed there. And I think it all goes back to when she was admitted and [Page 83] maybe as compiled by—complicated by other things even after that.

MR. BEASLEY: Including the frailties of the human mind—memory.

Q. (BY MR. ROGERS) On page 122 of Plaintiffs deposition, hospital's attorney, Mr. Beasley, states that the EKG was done within 35 minutes of arrival which—however, the hospital places Plaintiffs arrival at 1:02 and Plaintiff places her arrival at 12:30. So Plaintiff uses the lapse in time for the EKG as being 65 minutes *when* you add—if you start it from that point.

MR. BEASLEY: Shows it at 36 minutes by the way.

MR. ROGERS: Okay.

Q. (BY MR. ROGERS) Is that—I think that depending upon—this is a question I should ask you. It's one thing to do an EKG within 35 minutes for a person presenting with Plaintiffs symptoms. It's another thing to do an EKG 65 minutes, over an hour later.

MR. BEASLEY: Object to the [Page 84] form.

A. Okay.

Q. Do you think that that can be crucial in the outcomes, how quickly you begin doing diagnostic tests like EKGs because—because we have the start time, these dates are crucial. Plaintiff, her mother, and at some point her father are claiming that they actually looked at their watches, and the hospital's records are saying something differently. But would a 65-minute delay from the time of arrival at a hospital to the EKG being performed versus a 35-minute delay, is that significant or is it just not significant?

MR. BEASLEY: Form.

A. The question I guess is—or the answer is it depends. You know, what did the EKG show? What treatments were affected by that? It's hard to speak in generalities. I need to look at that in more of a specific patient—or a specific condition.

Q. Well, let's look at what the EKG did show. Well, let me ask you, what did this EKG show, assuming that it was done 65 [page 85] minutes later or—this is all on page 122 of Plaintiffs deposition by the way --whether it's done 65 minutes later or whether it's done 35 minutes later, the EKG results showed what?

MR BEASLEY: Object to the form. Repetitive.

A. It showed the ST segment elevation and the anterior leads.

Q. Okay. And elevated myoglobin levels. Was the myoglobin test done at that time?

MR. BEASLEY: EKG?

A. The myoglobin was probably available about 3:15, 3:20.

Q. Okay.

A. So the initial test was only the EKG.

Q. Okay. On page 123 of Plaintiffs deposition, the hospital's attorney states—that's Mr. Beasley—states that the hospital took x-rays within 50 minutes of Plaintiffs arrival; however, the hospital places Plaintiffs arrival time again at 1:02 p.m., but Plaintiff states that she arrived [page 110]

MR. ROGERS: Now, Dan, that was not good.

MR. BEASLEY: I don't mean to be derogatory but, here, look. There's his record. Have you read that? Darren Moore received her at 1920, admitted to CCU bed 20 from the ER. RN in attendance.

MR. ROGERS: Okay. We're going to work through that.

Q (BY MR. ROGERS) Now, Darren Moore, he said that he received the patient at—CCU, does that refer to the critical care unit?

A. Correct.

MR. BEASLEY: Right.

Q. At 7:20 p.m., correct?

MR. BEASLEY: Right

MR. ROGERS: Now, according to Dr. Chin, he hadn't even seen the Plaintiff for the cardiac catheterization. Plaintiff—the sequence of events was that the first thing that happened after Plaintiff left, if I'm not—you can chime in any time that you want to.

MR. BEASLEY: Well, let me chime in because this is where your client is so [page 111] confused. She went to the CCU unit from 7:20 until 8:00. At 8:00, she continued to complain of pain,. a six to ten level. Catheterization team arrived at 8:30. Sent to cath lab via stretcher by Darren Moore, and at 9:00, she returned from the cath lab.

MR ROGERS: Plaintiff was in the emergency room, correct?

MR. BEASLEY: Until 7:20.

MR. ROGERS: Of course we say it was—there's another discrepancy there, but we know that there's that discrepancy. Plaintiff, her parents put that time frame at 8:00 or 8:30.

MR. BEASLEY: I hear you.

Darren Moore will be one of the 35 who will say I don't know what she's talking about. MR ROGERS: Okay.

MR. BEASLEY: He was on duty that night in the CCU unit and nowhere else, and he saw her in his department at 7:20 at night.

MR. ROGERS: Okay. But that was still in the emergency room, correct?

[Page 112] MR. BEASLEY: No, sir. That was in the CCU unit on the CCU floor at 7:20 at night.

MR. ROGERS: Okay. What is

Dr. Chin saying then when he says that she was brought up to us in a fasting state?

MR. BEASLEY: She was at 8:00. MR. ROGERS: I mean from the emergency room, correct?

MR. BEASLEY: She was brought up to the cardiology department I guess is what he's talking about.

A. Again, I'm reading into it, but I think he's commenting that she was brought to the cath lab, which is separate from the CCU. So she was transferred from the emergency department to the CCU, and then she went from the CCU to the cath lab.

MR. BEASLEY: He didn't say brought from the ER. He said brought to the cath lab.

A. Yeah.

Q. (BY MR. ROGERS) And then he says that nine hours had elapsed, correct? Where is he getting this nine hours from?

[Page 113] MR. BEASLEY: You're going to have to ask him.

MR. ROGERS: I know, but the nine hours fits so. well into Plaintiffs—

MR. BEASLEY: He says nine hours after onset.

MR. ROGERS: Right. And the question is when was onset because he was relying upon I guess the records of Plaintiff saying that she had this sudden choking feeling.

MR. BEASLEY: Ask Dr. Andershock your question.

Q. (BY MR. ROGERS) Okay.

Dr.Andershock, Plaintiff did mention that the onset of her symptoms began a little before noon, correct? Do you remember reading that? It's in her history.

A. Yeah, I vaguely remember that.

Q. You vaguely remember that. And so in Dr. Chin's statements to Plaintiffs parents after the angioplasty, he, according to them in their depositions, describe exactly what I was going. on and states candidly that from the time that she left her house, this thing [Page 114] was a thing in progress. That's the thing that has me confused in terms of time line, and that's the reason why I was asking you questions concerning at what point did Dulan have a completely occluded left descending coronary artery?

MR. BEASLEY: Is that question *at what time* did she have a completely occluded—

MR. ROGERS: Occluded coronary descending artery.

MR. BEASLEY: I think it's been asked and answered, but go ahead.

A. Again, the answer to. that is I don't know.

Q. Okay. Is it possible—I mean is it possible that it was occluded and blocked from the time that she presented herself to the emergency room?

A. Yes.

Q. Is it likely that—I mean I know I'm asking you these--is it likely that it was completely occluded or do you <u>think</u> it just became occluded during the stretch that she was in the hospital?

[Page 115] MR. BEASLEY: Object to the form. Lack of predicate.

A. I don't know. All I can say is that her EKG certainly changed from the first one to the second one and even to the third one, so there seemed to be some process in evolution. In terms of when it exactly occurred, I do not know.

Q. Okay. Are you saying that the first one was the first one was abnormal

A. Uh-huh.

Q. And then the second one was you're saying the evolution of the test. Now, was the second EKG abnormal also?

A. Yes. They're all abnormal, but there are-changes to each specific EKG.

Q Were they getting better or would you say they were getting worse?

A. Well, I mean we have Dr: Chin's interpretation—official interpretation already, and on the first two—I mean the first one—

Q. Is there a time frame?

A. You know, he said ST elevation, consider early replolarization, pericarditis,[page 116] or injury, or even possible the digitalis effect. That's another one to put in there. I'm sorry, that was the second one.

MR BEASLEY: No.

A. No, that was the first one. The second one was ST elevation, consider early repolarization, pericarditis or injury. Nonspecific T wave, abnormal ECG. So there are changes that you can see between the two that he commented on.

Q. Now—

A. When exactly she had—I don't know.

Q. Okay. This is—we're really winding it down now. One thing that I'm not sure of is at what point was Dr. Chin involved in this process. It appears as though that he's acting surprised, according to Plaintiff, when she sees—as per her affidavit, upon initially seeing her in the cath lab, he states—

MR BEASLEY: Object to the form.

Q. He states, according to Plaintiffs deposition, I can't believe they left you [page 117] down there for almost nine hours or for nine hours or however many he said, but he used the word nine. And my question is because he sounds so shocked and surprised, why is he sounding shocked and surprised if in fact he was involved much earlier?

MR. BEASLEY: Object to the form.

Q. When was he involved?

MR. BEASLEY Wait a minute. Is the question when was Chin involved?

Q. (BY MR ROGERS) When was Chin involved? Let's start there.

A. It would have been helpful if he had recorded some of these comments in the record or his thought process, but he didn't so it's difficult to speak to his mindset.

MR. BEASLEY: Well, let's just ask—the question is when did he get involved?

A. When he got involved, he was actually called by Dr. Gessler so I had handed over care to Dr. Dulan to Dr. Gessler. She was sent up to the—it's back here. Sent up to the CCU, and from there the decision was made [page 118] to send her to the cath lab apparently about 8:00 is when I see is when the cath lab team is called. That would be about the time he would be contacted. Now, I was not involved with that. Dr. Gessler contacted Dr. Chin and asked him to come in.

Q. Okay. Again, the reason for my confusion, Dr. Andershock, is that the Plaintiff or her parents are steadfastly claiming in some instances to actually looking at watches and saying that this time was—there's no question that it was this time as opposed to the time that the hospital put in their records. And then what caps it off is that Dr. Chin makes statements that can only be verified on a credibility basis by the Plaintiff who claims that when she was wheeled into the cath lab, he said I can't believe that they left you down there for almost nine hours.

So the use of the term nine hours, I just start wondering, well, where are we measuring that from because if—let's just stop and think about it. If in fact we use the hospital's time of one hour—1:02 or [page 119] 1:03, if you add nine hours to that, it would be 10:00. If you add nine hours to Plaintiffs

version of onset, then it would be almost nine hours maybe by the time she got up to catheterization lab, maybe it would be a little—it would be a quarter to nine or something like that, which would be consistent with Plaintiffs time line. And so I'm confused.

MR. BEASLEY: Well, as I recall, the parents didn't talk to Chin until after they were through with the cath lab procedure—

MR. ROGERS: That is correct.

MR. BEASLEY:'—which would have been close to 10:00 at night when he wrote his progress note. And, you know, the nine hours could easily have come from Dr. Dulan. He said how long have you been here at the hospital or when did it start, and she could have said nine hours ago. I mean—

MR. ROGERS: But I don't think she had ever talked to him before—

MR. BEASLEY:—at one point [page 120] when she first came, she said it began this morning.

MR. ROGERS: Right.

MR. BEASLEY: She told

Dr. Andershock at 1:00. She told another nurse 45 minutes ago, and that was at 12:30. So there's five different times in there.

MR. ROGERS: Well, I remember in your deposition, you were mentioning because she mentioned several hours had elapsed and then you were stringing together a number of hours and saying what are you saying in terms of several hours, and then it became clear to me what she was trying to say. She was using several hours based upon a 12:30 time frame and then several hours elapsing from that point as opposed to several hours from—

MR. BEASLEY: I just don't think Dr. Andershock can really explain to you what—

MR. ROGERS: I believe you're correct.

MR. BEASLEY:—Dr. Chin meant or what the basis of his time frame was. He doesn't know where he got that time.

[page 121] Q. (BY MR ROGERS) Dr. Chin—was he involved during the time when—let's say at 3:00 in the afternoon, was he involved then.

A. No.

Q. Was he involved for the very first time—

A. No.

Q. when she was wheeled up to the catheterization lab?

A. Yes. Yes.

Q. And so unless the Plaintiff is just not being truthful, she says that upon seeing her at the cath lab, he exclaims to her I can't believe they left you down there for nine hours, and that is what's causing me and because it jives with her time frame and because—to be honest with you,

Dr. Andershock, what's puzzling me is that other medical professionals, including well respected—at least one well-respected cardiologist, and I need to turn over my expert witnesses to you very soon, we're still working on that, has said that the records just don't make any sense to him.

A. Right.

[Page 122] Q. And so if this was just one doctor saying that, then I would say, well, maybe you just don't understand the records. But when it's more than one doctor, you know, I like to get to the heart of it.

MR. BEASLEY: Okay. What's the question?

Q. (BY MR. ROGERS) The question is can you explain Dr. Chin's time line? It's the last time going to ask you.

MR. BEASLEY: Which statement exactly are you referring to? Which statement by Dr. Chin are you referring to in the record?

MR ROGERS: Okay. I'm going to refer to all of them. The one in the affidavit, which is at the bottom of—

MR. BEASLEY: Dr. Chin's time line. He says she was already nine hours after onset of symptoms. Is that what you're referring to?

MR. ROGERS: Precisely.

Q. (BY MR. ROGERS) What are the symptoms that he's talking about? Also—

MR. BEASLEY: Can you provide [page 123] any insights or elucidations as to where Chin made that statement or the basis of how he made that statement?

A. I can speculate if you would like me to. I assume he's talking about the chest pain.

Q. Well, I guess—now, and we've already asked. We've already kind of explored this whether it's possible for her to have actually had the completely occluded left descending coronary artery as of 12 noon. You're saying it's a possibility that it could have been completely occluded as of 12 noon. In other words, Dr: Chin ultimately diagnoses her as having a completely occluded left descending coronary artery, and I guess the question is could Plaintiff have left —potentially left her house in that condition

MR. BEASLEY: Object –

Q.—at 12 noon?

MR. BEASLEY: Object to the form.

A. The answer to that is, again, I don't know. It's possible she may have had a [page 124] stuttering occlusion. She may have occluded at some point after she presented to the hospital. It's difficult to say. Dr. Chin has, you know, obviously the benefit of having done the heart catheterization and knowing that it was completely occluded. **In** terms of when it occluded, I could not tell you.

Q. So it's theoretically possible at least that when Plaintiff left her house that she could have been full-blown in a heart attack?

MR. BEASLEY: Object to the form.

Q. It's possible?

A. It's possible, yes.

Q. And, again, under oath, Plaintiff states in paragraph 28 of her affidavit starting with—let me just do 27. I was taken from the emergency room—and that's Plaintiff's thinking. I was taken from the emergency room and admitted to the hospital for a heart catheterization procedure. That's at least her—and what I hear you saying that when they took her—admitted [page 125] her to the hospital, they admitted her to CCU—

A. Correct.

Q. first? And then there was some time lapse there, maybe not long, because they wanted to hurry up and do this heart catheterization procedure I assume. But then in any event, number 28, Dr. Chin, the cardiologist assigned to my case, upon seeing me stated I can't believe they left you down there for almost nine hours. She says I agree it was a traumatic experience.

The thing that I'm trying to determine—which you've done as well as you can for me today, Dr. Andershock. I understand that you can't help me any more on this is the fact that he does repeat nine hours in his report. So it's something that's in his mind, and because the nine hours jives with

Plaintiff's time line, it becomes an unresolved issue as to whose account is accurate.

MR. BEASLEY: Is there a question?

MR. ROGERS: No. There's a

APPENDIX I

[174] when they moved me into the larger room, they attempted, in order to reduce the pain. The point is, nothing they did worked. Nothing.

Q So, you're not saying this is the first time you had had Nitroglycerine put on your tongue?

A No, I'm not saying it.

Q I say that because according to the chart--

A I'm not saying it's the first time.

Q. According to the chart, you received two baby aspirins, nitroglycerine paste, and sublingual Nitroglycerine at 3:10 that afternoon?

A I think that's probably correct.

Q Do you recall any explanation by the nursing staff as to why you were being given those medications?

A No explanation was given.

Q Were you told you were given Nitroglycerine?

APPENDIX J

[277] indicators of treatment in the emergency room indicate that there's no statistically significant difference between the treatment of whites and blacks; and if you have other indicators that indicate that blacks received more favorable treatment than whites and other indicators, or an indicator, that whites received more favorable treatment than blacks; does that present a picture from your view as a statistician of an emergency room or group of emergency room personnel or physicians in the emergency room had an intention to discriminate in favor of Caucasians against African-Americans?

A. I can't speculate what their intentions were. I can say that the only statistically significant finding showed that African-Americans were less likely to be administered aspirin than Caucasians.

Q. And there were—

A. The only statistically significant finding showed African-Americans to receive less of something. That's what the data showed.

Q. Aspirin, right?

APPENDIX K

American women have a 13 percent greater chance of suffering from a heart attack than white women?

MR. BEASLEY: Object to the form.

A. That specific number, no, I was not aware of.

Q. Okay. Were you familiar on October 6, 2000, to kind of pin down a date, with studies that state that delaying angioplasty beyond two hours for persons experiencing a heart attack increases the chance of death by 40 percent to 63 percent?

MR. BEASLEY: Object. to. the form.

A. No.

Q. You were familiar, however, with studies that indicated that-postmenopausal women, such as Plaintiff, are at greater risk for a heart attack?.

MR. BEASLEY: Object to the form

A. Yes.

Q. Dr. Andershock, did you obtain Plaintiffs myoglobin level? And I hope I pronounced that right.

A. Yes, as part of the cardiac panel

Q. Was. Plaintiffs myoglobin level normal or acceptable?

A. It showed a level of 75.4 from the record, and the upper limit of normal is 58.

Q. Could you interpret that? I'm. not sure exactly are you saying that was abnormal?

A. It was elevated.

Q. It was elevated What is the significance of an elevated myoglobin level? When physicians see an elevated myoglobin level, does that send some sort of message to physicians?

MR. BEASLEY: Object to the form.

A. Myoglobin is present in skeletal muscle, cardiac muscle and smooth muscle, and there are literally hundreds of conditions that can cause an elevated myoglobin. So in the setting of an elevated myoglobin, you have to take it in conjunction with the clinical correlation. Specifically, it is a very poor marker for cardiac ischemia as a stand-alone test.

Q. Let me ask you this then. If Plaintiff—if an emergency room then bad in its possession reports indicating that a person had an elevated myoglobin level, that the person's initial EKG was abnormal, that the person-was 15 years postmenopausal, that the person, in this case the Plaintiff, that the person. had a history of hypertension, that the person was experiencing substernal chest pain that radiates to her neck and shoulder and shortness of breath, might that cause concern beyond just further testing?

MR BEASLEY: Object to the form.

A. Let me see if I understand your question correctly. You're asking about the interpretation of the myoglobin in conjunction to—

Q. With all

A. her clinical

Q. Right.

A. To her specific clinical presentation?

Q. Yes. Dr. Andershock, I guess what

I'm really asking is were the stars just lined up just so right that it might be reasonable to say we need to do a heart imaging test?

MR. BEASLEY: Object to the form. What are you calling heart imaging?

Q. (BY MR. ROGERS) I'm sorry. That's—heart imaging test is this layman's reference to a heart catheterization.

A. Okay.

MR. BEASLEY: Object to the form.

A. I have two answers to that question.. The first part is that with cardiac enzymes, if they're negative, and I can—and most places now do not even use myoglobin as part of their cardiac panel. That's been dropped. They just use troponin and CK or CKMB. In fact, if you go through her hospitalization,, myoglobin doesn't show up anymore. They just check CKMB and troponins. So that really is not a test that we would hang our hat on. In the setting of negative cardiac enzymes, if they have a strong clinical